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PNEUMONECTOMY FOR SEVERE IRRADIATION DAMAGE OF THE LUNG

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ALTHOUGH thirty years have passed since Groover, Christie, and Merritt[1] first called attention to the syndrome of lung damage as a result of exposure to ionizing irradiation, no progress has been made in the treatment of this disease. In fact, a review of the literature reveals a virtually complete lack of consideration of this phase of the problem despite extensive preoccupation with the clinical and pathologic aspects of the syndrome. Since the first successful pneumonectomy for carcinoma was done in 1933,[2] vast progress has been made in thoracic surgery, and many conditions previously regarded as incurable have yielded to the surgical attack. When a patient with lung damage due to roentgen irradiation presented herself to us in 1947, it appeared logical to explore the question of whether this might not be one more condition amenable to successful correction by surgery.

Many case reports and experimental studies have been published since the original identification of the lesion, and this extensive literature has been reviewed by Desjardins,[3] Engelstad,[4] and more recently by Warren.[5] While a comprehensive review of the literature is, therefore, unnecessary at this time, a summary of the accumulated knowledge of the effect of irradiation on the lung is in order.

EFFECT OF IONIZING IRRADIATION ON THE LUNGS OF EXPERIMENTAL ANIMALS

Of the many experimental studies, only three will be considered here because they are representative and because the work to be discussed is of outstanding significance.

Lüdin and Werthemann's work,[6] although carried out on small numbers of animals, produced significant results. Their first series of experiments consisted of five rabbits given 500 r. of roentgen irradiation measured in air to alternate anterior and posterior chest fields measuring 5 by 5 centimeters. One treatment

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was given weekly until the animals died of the effects. From seventeen to forty-three treatments were administered. The changes produced consisted of increase in consistency of the lungs and production of pleural adhesions. Some parts of the lungs showed diffuse pneumonic infiltrations, others showed multiple small, firm, nodular foci of increased density. Many of the bronchi contained thick pus which was sterile on culture. In the least affected lungs the microscopic picture was that of patchy areas of atelectasis, and scattered alveoli with thickened walls. The alveolar capillaries were congested, and scattered alveoli contained edema fluid. In some alveoli degenerative changes in the lining cells were observed. The mildest changes in the bronchi consisted of hypersecretion of mucus and degeneration of the epithelium. In the severely affected areas purulent bronchitis, bronchiolitis, and bronchopneumonia occurred. This was accompanied by extensive destruction of alveolar epithelium and regeneration of the bronchial epithelium, as evidenced by the presence of scattered mitoses. The second series consisted of six rabbits which were given the same amount of irradiation, but the authors used 12 by 13 centimeter fields. This increased the amount of irradiation reaching the lung by an increase in the scattered irradiation. As a result the animals succumbed much earlier than those of the first series, so that only fourteen to fifteen weekly treatments were given. The grossly visible changes in these lungs were much more marked. Usually all parts of the lungs were affected, and individual foci could not be distinguished. The bronchitic, bronchiolitic, and bronchopneumonic processes observed in the more heavily irradiated animals of the first series were again seen. In addition, all the animals showed epithelial proliferations of varying degree. The bronchial epithelium became tall and stratified, and hypersecretion of mucin was marked. The most striking epithelial changes were seen in the peribronchial lung parenchyma. Alveolar structures lined by cuboidal epithelium are described and photographed, and are interpreted as arising from the bronchial epithelium. These authors believed these structures to result from infiltration of peribronchial tissue by proliferating bronchial epithelium which is thus regarded as showing autonomous growth characteristics. A study of the photomicrographs, however, suggests that these structures might represent peribronchial alveoli whose epithelium is very hyperplastic. Nevertheless, the concept that neoplasia might be induced by irradiation cannot be completely rejected. The frequent occurrence of bronchiogenic carcinoma in the Schneeberg miners is well known, and these growths may be instances of neoplasms induced by ionizing rays.

Engelstad's experiments[4] are the most extensive on record. One hundred sixteen rabbits were examined of which one hundred one had received roentgen irradiation to the chest, while fifteen served as controls. It was shown that different types of irradiation did not produce qualitative or quantitative differences in the effect on the lungs, provided that the total tissue dose and intensity of irradiation remained constant. Total dose, and the time factor, i.e., the intensity of irradiation, played a marked role in determining the severity of the reaction which became less with fractionation and protraction of a given dose of irradiation. The quantities of irradiation used in these experiments were

based on the skin changes produced. Three types of irradiation were employed: subepidermicidal doses which provoked slight or no skin reactions, epidermicidal doses which produced an epidermitis lasting up to six weeks, and dangerous or lethal doses which caused severe skin reactions in all animals and led to death within a short time in many. The total dose of irradiation was given in the course of one day or in a few days, at most twelve.

After subepidermicidal doses lung changes were slight and recovery virtually complete within two months, leaving in its wake a mild fibrosis. Epidermicidal doses caused a rather severe and typical reaction which could be divided into four stages. The initial stage coincided in time with the early erythema of the skin, and consisted of degeneration of lymph follicles, hypersecretion of bronchial mucus, and hyperemia and leucocytic infiltration. The second stage, a latent period, lasted two to three weeks. After this the main reaction occurred. It was characterized by degenerative changes in the bronchial epithelium and in the pulmonary stroma and by various degrees of acute inflammation in the form of peribronchial, perivascular, and bronchopneumonic infiltrations. The bronchial cartilage proved to be relatively radioresistant. Alveolar macrophages, occasionally bizarre and multinucleated, were common. The last stage of the reaction began after the subsidence of the main reaction, usually after one to two months. Regenerative and reparative processes dominated this phase. Its main features were proliferation of connective tissue, sclerosis, and proliferative changes in the bronchial epithelium, and more rarely in the alveolar epithelium. These processes were not complete at the end of six months.

After dangerous or lethal doses the reaction differed from the above only in being more severe and in that the sclerosing processes of the reparative stage were more exuberant. Ossification of portions of the lungs was common. Thus Engelstad demonstrated a direct relationship between the amount of irradiation and its effect on the lung. He showed that small doses produce only mild or insignificant changes, and that the significant reaction caused by larger doses consists of an early acute inflammatory reaction and a prolonged late sclerosing process.

Warren and Gates[7] contributed significantly to our understanding of the pathology of the experimental radiation reaction by an exact histologic analysis of the mild early effects of irradiation on the lungs of rats, rabbits, dogs, and pigs. The earliest changes in the alveolar and atrial lining cells were increase in cell size and nuclear hypertrophy, often with clumping of chromatin, and the appearance of one or more large nucleoli. Lack of uniformity of this change from one alveolus to another was characteristic of this reaction. The appearance of a hyaline membrane was infrequent in animals, and this structure will be discussed more fully in the section on the pathology of the irradiation reaction of the human lung. The alveolar walls were affected by an irregularly distributed edema and congestion. Increased cellularity and, if the reaction were more pronounced, thickening and splitting of the elastic fibers were found. More severe changes of fibrosis and hyalinization were not seen because only relatively small doses of irradiation were administered. Bronchial epithelium

showed only an increased number of mucous cells and a change of the columnar ciliated cells to cuboid nonciliated forms. Bronchiolar epithelium approached alveolar cells in sensitivity, and similar anaplastic changes were seen. Cartilage, smooth muscle, and connective tissue showed no clear-cut changes. Blood vessels, lymphatics, supporting connective tissue septa, and pleura showed only mild changes, consisting of edema and congestion early in the reaction and mild fibrosis in the later stages.

Quantities of irradiation well within the therapeutic range thus cause reactions which must unquestionably severely disturb pulmonary function and increase the susceptibility of the lung to secondary infection.

THE CLINICAL PICTURE OF RADIATION DAMAGE OF THE LUNG

Roentgen irradiation of the human chest is almost always carried out for the treatment of a malignant neoplasm. Such neoplasms may be intrathoracic, e.g., mediastinal lymphomas, eosphageal carcinoma, inoperable bronchiogenic carcinoma, but the commonest indication for roentgen treatments of the chest is carcinoma of the breast. A discussion of the place of roentgen therapy in the treatment of breast cancer is outside the scope of this paper, and we do not feel qualified to discuss methods of administration of roentgen rays. However, regardless of the method used, a certain amount of the irradiation will reach the underlying lung, and often the effects of pulmonary irradiation will become clinically significant. The frequency with which this occurs is variable. Engelstad[8] encountered lung changes in 5.4 per cent of 386 patients who were given roentgen therapy for breast cancer, and in 20 per cent of forty-nine cases treated for carcinoma of the esophagus. These figures are in agreement with those of Warren and Spencer,[9] who demonstrated histologic changes due to radiation in 12 per cent of 234 patients treated for a variety of causes.

The group of patients who receive roentgen therapy for breast cancer should be especially emphasized, not only because they constitute the largest single group, but also because radiation in these cases is usually confined to one hemithorax and because their chances for long-term survival are better than those of most patients with intrathoracic lesions who receive roentgen therapy. Characteristically, the clinical history is that a middle-aged or elderly woman with a recent radical mastectomy is given postoperative x-ray treatments. While the patient is still receiving therapy or three to six weeks later a dry or moderately productive cough begins. If the dose of irradiation is moderate (less than 130 to 140 per cent of the skin unit dose according to Evans and Leucutia[10]) the cough may persist for several months and eventually disappear. If greater quantities of irradiation reached the lung, the cough may become progressively worse. In some instances the cough is not noticed until several months have passed. The quantities of sputum produced may vary. Dyspnea is a very common symptom, and may be paroxysmal and disabling. Chest pain is a frequent complaint, but hemoptysis is relatively uncommon. Many of these patients are in good general health, and this finding may aid in the clinical differentiation of the irradiation reaction from pulmonary metastases. The

physical signs of pulmonary irradiation damage are variable. Fever is usually absent, but may occur when secondary infection is superimposed on the irradiation reaction. The affected hemithorax usually shows skin changes such as scaling, pigmentation, and induration, and respiratory excursions are much diminished. Examination of the lung may show areas of impaired percussion note with râles and bronchial breathing. In other areas, especially late in the disease, breath sounds, voice sounds, and fremitus may be diminished or absent. In the early stages of the reaction the only findings may be scattered râles and insignificant changes in the breath sounds.

THE RADIOLOGIC PICTURE OF PULMONARY IRRADIATION DAMAGE

The earliest radiologic manifestation of pulmonary irradiation damage is said by Warren and Spencer[9] to be an increase in radiolucency of the involved lung. Other observers[11] describe a diffuse haziness as the earliest sign. With progression of the lesion, irregular patchy consolidations radiating out from the hilum appear. Pleural reaction and adhesions, and occasionally pleural effusions may become visible. The late changes are dominated by a picture of pulmonary sclerosis and shrinkage. The lung is atelectatic, the trachea and mediastinal structures shift toward the affected side, and the interspaces become narrow. Bronchiectatic cavities may be visible,[12] and in the lateral view the changes may in some instances be notably more severe anteriorly where most of the irradiation was directed. Various linear and nodular shadows have been described, but it is generally agreed that there is no single pattern pathognomonic of the disease.

Clinically and radiologically the lesion must be differentiated from various types of pneumonitis, nonspecific bronchiectasis, tuberculosis, and metastatic tumor. Of course, radiation damage and metastases can and frequently do coexist. However, in most cases it should be possible to identify the radiation reaction by correlating the clinical history and the roentgenologic findings.

THE PATHOLOGY OF PULMONARY IRRADIATION DAMAGE IN MAN

Knowledge of the pathology of irradiation effect on the human lung has been based so far entirely on autopsy studies. The changes described are largely comparable to those produced in experimental animals and outlined above. Thus, Belt[13] and Toenges and Kalbfleisch,[14] in two cases of radiation damage due to occupational inhalation of radioactive dust, and Schairer and Krombach,[15] in a case of fibrosis due to roentgen therapy, described gross changes in the lung consisting of marked diminution in size and increase in consistency. The microscopic changes observed were extensive fibrosis and atelectasis. Alveolar walls were thickened and contained increased numbers of coarse elastic fibers. Many of the alveoli were filled with edema fluid and macrophages, and some alveolar lining cells showed degeneration. In Schairer and Krombach's[15] case marked epithelial hyperplasia was found. Interlobular septa were thickened and fibrotic. In the bronchi mild dilatation of the medium and smaller bronchi and mild epithelial changes were observed. Occasional bronchial cartilages were calcified. In two of these cases[13, 15] a hyaline eosinophilic membrane lining many

of the alveoli was observed. This membrane was similar to that described by Farber and Wilson.[16] It is known to occur in influenza, plague, neonatal pneumonia, and mustard gas poisoning. More recently Warren and Gates[7] have again emphasized this finding. They state that in their material the reaction in human lungs is entirely comparable to that seen in experimental animals with the following qualifications. In man the alveolar epithelial changes tend to be more marked than in animals, and bronchial epithelial changes are less prominent. In the human cases the presence of hyaline membranes was a rather constant finding. Warren and Spencer[9] state that the membrane was virtually always found in patients who had definite pulmonary irradiation damage. The combined finding of hyaline membranes, epithelial anaplasia, and rupture and reduplication of the elastica were considered diagnostic of the disease.

THE PATHOLOGIC PHYSIOLOGY OF PULMONARY IRRADIATION DAMAGE

The changes in lung function resulting from the alterations in pulmonary structure have not yet been completely clarified. However, from the investigations of Leach,[17] and Baldwin, Cournand and Richards[18] the following considerations emerge. Patients with radiation damage of one lung show a marked restriction in vital capacity. Compared with normal control subjects, their complementary and supplementary air values are decreased by about one-half. Their maximum breathing capacity is comparably reduced. At rest their ventilation is normal or moderately increased, but with exercise marked hyperventilation occurs. These two factors, hyperventilation and reduced maximum breathing capacity, result in a marked diminution of breathing reserve to as low as 40 per cent. Dyspnea usually accompanies such greatly lowered ventilatory reserve. One can, of course, explain the reduction in vital capacity and maximum breathing capacity on the basis of the anatomic findings. The relatively immobile hemithorax, the extensive replacement of pulmonary parenchyma by scar tissue, and the accompanying inflammatory changes adequately account for the severe ventilatory insufficiency. The cause of the hyperventilation, however, is more obscure. It has been shown that arterial oxygen saturation at rest and during exercise is normal in patients with radiation damage. Similarly, alterations in plasma carbon dioxide content have not been observed. Bronchospirometric studies have shown that this is due primarily to continued satisfactory function by the uninvolved lung. One must, therefore, conclude that patients with irradiation damage of one lung do not hyperventilate because of a necessity to restore to physiologic equilibrium an abnormal blood chemical condition. In fact, they appear to hyperventilate despite the absence of any known physiologic need for such activity. For these reasons hyperventilation and the resultant dyspnea are believed to be caused by abnormal reflexes originating in the altered lung.

THE THERAPY OF UNILATERAL PULMONARY IRRADIATION DAMAGE

After consideration of all the known facts about the pathology and physiology of unilateral radiation damage of the human lung, it seemed logical to

attack this therapeutic problem in a more direct and effective manner than had hitherto been attempted. If, as we believe, the dyspnea of the unilateral pulmonary irradiation reaction is caused by abnormal reflexes arising in the irradiated lung, and if the structural changes fully account for the other symptoms such as cough, sputum, chest pain, and occasional hemoptyses, then the logical treatment of unilateral radiation damage would seem to be to rid the patient of the diseased lung by pneumonectomy. To date we have had occasion to carry out this plan of therapy in two patients.

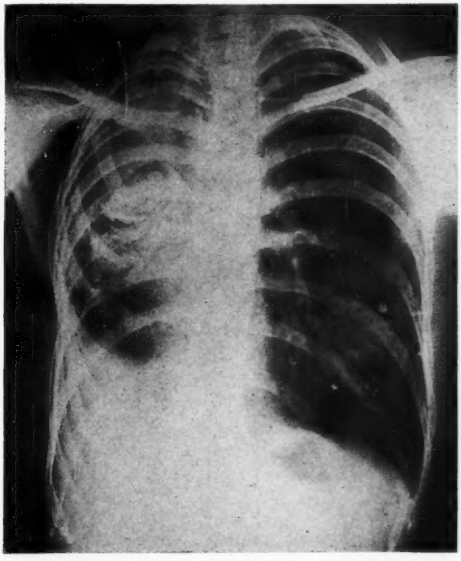

Fig. 1.—Case 1. Radiograph of the chest. The right lung is atelectatic and contains residual lipiodol. The mediastinum is shifted to the right and there is compensatory emphysema of the left lung.

CASE REPORTS

Case 1.—M. J. K. (History No. 154012). This 42-year-old white unmarried woman was first seen at Barnes Hospital on Dec. 16, 1947, because of cough and dyspnea. Twenty-two months before admission, in February, 1945, the patient had undergone elsewhere a right radical mastectomy for carcinoma of the breast with axillary lymph node metastases. During the next four months she received two courses of roentgen therapy to the right chest and axilla, and a third course was given in December, 1946. Thereafter, she developed a chronic cough productive of small amounts of watery sputum, and became dyspneic. In February, 1947, she was noted to have a pleural effusion which was withdrawn every three weeks until July. There had been no hemoptysis or chest pain. The patient's weight had not changed significantly. Physical examination showed a thin white woman who was in no obvious respiratory distress. The vital signs were normal. The significant findings were limited to the neck and chest. The trachea was deviated to the right side. There was asymmetry and reduction in size of the right thorax. The right breast and pectoral muscles were absent, and there was an old surgical scar. The scar was tender, and the surrounding skin showed a marked irradiation effect. Percussion note over the right chest was dull, and

breath sounds were harsh. The left outer cardiac dullness was displaced toward the midline. There was no evidence of any recurrence of the carcinoma of the breast. Roentgenogram of the chest (Fig. 1) showed displacement of the mediastinal structures into the right chest and a scoliosis of the dorsal spine with the concavity to the right. The left lung was emphysematous. Only a small amount of aerated lung was seen on the right. The rest of the right hemithorax was obscured by a homogeneous density. The intercostal spaces on the right were narrowed. All laboratory studies were within normal limits, and oximetric studies showed normal arterial oxygen saturation with no abnormalities in response to exercise. Because of this finding further observation was decided on and the patient was discharged on Dec. 24, 1947.

Fig. 2.

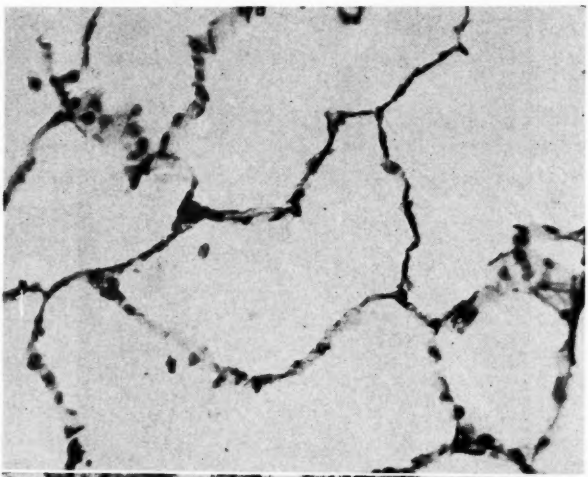

Fig. 3.

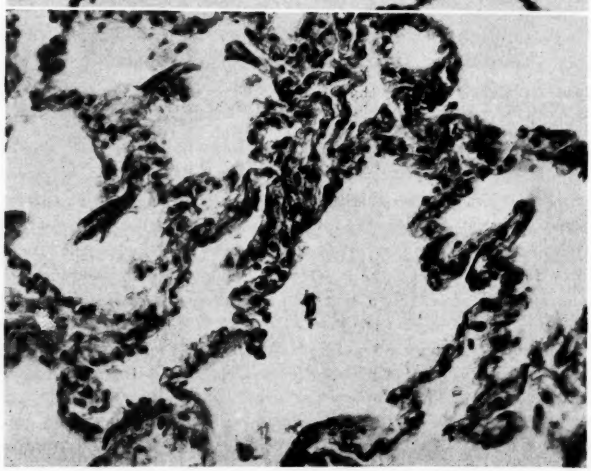

Fig. 2.—Photomicrograph of a normal lung stained for elastic tissue. Only occasional elastic (black) fibers are visible. (Verhoeff van Gieson stain ×400.)

Fig. 3.—Photomicrograph of a lung with alveolar thickening due to tuberculosis. There is no appreciable increase in the elastic tissue. (Verhoeff van Gieson stain ×400.)

The patient was readmitted to the hospital on June 10, 1948, because of persistent cough and dyspnea. For three months prior to admission this had required 100 mg. of demerol nightly for relief. The physical findings were unchanged from the previous admission. The roentgenologic findings remained unchanged. It was decided to attempt bronchospirometry, but at bronchoscopy the right tracheobronchial tree continually filled with thin watery secretions, and the patient became practically pulseless and dyspneic. The bronchoscope was, therefore, withdrawn, and no studies were done. Right pneumonectomy was carried out on June 14, 1948. The operation was technically difficult because of extensive pleural adhesions. The pleura was much thickened, and the lung was small. The patient's

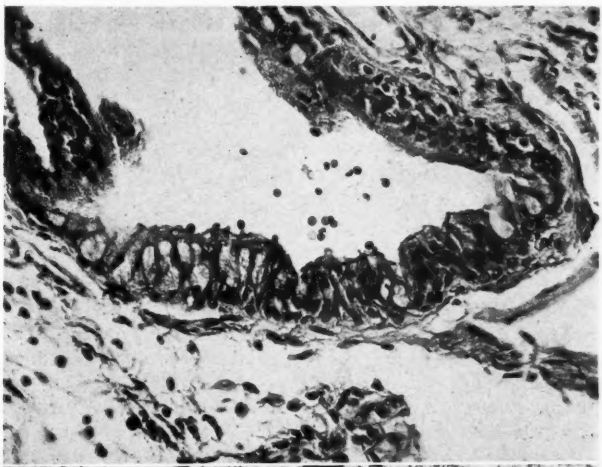

Fig. 4.

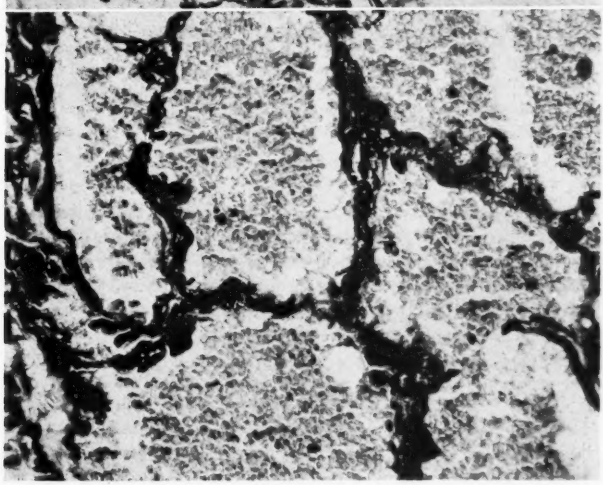

Fig. 5.

Fig. 4.—Case 1. Photomicrograph of one of the medium-sized bronchi to demonstrate increase in goblet cells of the bronchial mucosa. (Mucicarmine stain ×400.)

Fig. 5.—Case 1. Photomicrograph of pulmonary alveoli to show marked increase in number and size of elastic fibers. (Verhoeff van Gieson stain ×400.)

postoperative course was uneventful, and she was discharged on July 12, 1948. At the time of discharge her cough, although still present, was much improved. The patient's third admission to Barnes Hospital was from Feb. 16, 1949, to Feb. 19, 1949. At that time the patient stated that following her last hospitalization she had had much less cough and dyspnea and had felt well. However, since December, 1948, she had experienced several episodes of paroxysmal coughing and dyspnea. Physical examination was uninformative. The remaining left lung was clear. External spirometry showed a vital capacity of 1,300 c.c. Arterial oxygen saturation was 99 per cent at rest and 94 per cent after exercise. The patient was reassured about her condition and discharged.

The patient returned for the fourth time on April 26, 1950. Following her last admission the patient had continued to improve. Respiratory function studies in December, 1949, had shown a vital capacity of 1,450 c.c., total lung volume of 2,500 c.c., and maximum breathing capacity of 39 liters per minute. Arterial oxygen saturation at rest and after exercise was 100 per cent, and 21.3 liters were ventilated during the seventh minute of exercise. She tolerated exercise well and was considered much improved over her preoperative condition. Shortly after that she began to cough up large amounts of bloody sputum, developed chills and fever, and lost weight. Thoracentesis on the right side demonstrated the presence of an empyema. Thoracostomy drainage was instituted on April 27, 1950, and the patient was discharged greatly improved on May 2, 1950.

She returned to Barnes Hospital on Sept. 6, 1950, for a thoracoplasty to obliterate a residual empyema cavity. Except for a mild anemia her condition was good, and she tolerated the operation well. Her postoperative course was uneventful and she was discharged on Sept. 30, 1950, with only a small granulating cavity remaining.

Gross description of pathologic specimen (Surgical Pathology No. 61650): The specimen consisted of a small atelectatic lung. The visceral pleura was thickened, and posteriorly parietal pleura, also much thickened, was adherent to the lung. The bronchi were not remarkable. The upper lobe was completely atelectatic, hard, firm, and reddish brown. In the midportion of the lung considerable fibrosis was visible.

*Microscopic description:** Sections from various areas showed the lung to be severely damaged in all parts. The large bronchi were lined by a rather low columnar ciliated epithelium with scattered areas of squamous metaplasia of moderate degree. The bronchial walls were slightly edematous. The larger cartilage plates were not abnormal. In the smaller bronchi and bronchioles the changes were more pronounced. There was an increase in the number of mucin producing cells in the epithelium (Fig. 4). The lumen was dilated, and the smaller cartilages showed definite evidence of degeneration, especially around their periphery. The cartilage cells were very variable.

Vascular changes included mild edema of the vessel walls, and subintimal proliferation of connective tissue. The alveoli were preserved in only scattered areas. For the most part, they were replaced by dense fibrous tissue rich in thick elastic fibers. Where individual alveoli remained their walls were thickened and contained increased numbers of elastic tissue (Fig. 5). They were lined by a low cuboidal epithelium whose cells were variable in size and occasionally multinucleated. Their lumen contained blood, many macrophages, and occasional inflammatory cells. The pleura was thickened and somewhat edematous.

CASE 2.—S. B. L. (History No. 189570). This 45-year-old white married woman entered Barnes Hospital on Oct. 2, 1950, because of a harassing cough for about one year. In August, 1949, two weeks after she discovered a small lump in her right breast, she underwent a right radical mastectomy in another hospital. It is not known whether regional lymph nodes were involved. Her postoperative hospital course was uneventful. Beginning on the eighth post-

*The microscopic slides of this and the following case were reviewed by Dr. Lauren V. Ackerman.

operative day she received four daily x-ray treatments. After two weeks she was given sixteen more daily treatments to the anterior chest, the axillary region, and the supraclavicular area. She developed a severe skin reaction which required daily dressings for a long time. About eight weeks after her operation she developed a post-transfusion hepatitis for which she was hospitalized for five weeks. Soon after she left the hospital, about two months after the last x-ray treatment, she first noted a mild irritating nonproductive cough. In about three weeks she again became ill, this time with a pneumonia with severe cough productive of about a cup of mucoid sputum daily. She was treated with antibiotics which reduced her fever, but the cough persisted and became worse. At the time of this admission she complained of violent paroxysms of coughing resulting in vomiting. Exercise and talking appeared to bring on these paroxysms. Her cough was still productive of about a cup of mucoid sputum. There

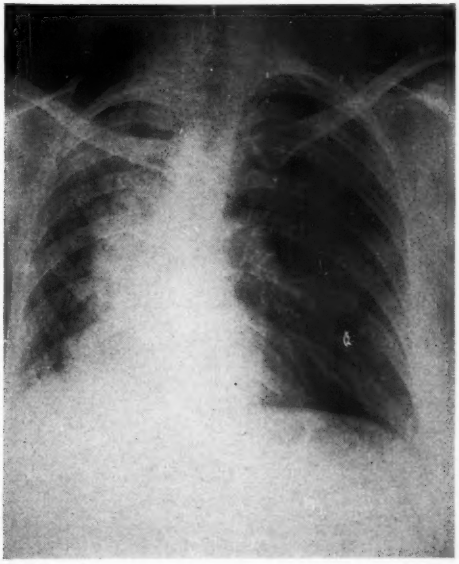

Fig. 6.—Case 2. Radiograph of the chest. The right lung is atelectatic and the mediastinal structures are shifted to the right.

had been no hemoptysis, but she had noticed mild dyspnea on exertion in recent months. On physical examination the vital signs were normal. The patient was well nourished. During the examination she was seized with frequent severe paroxysms of coughing. The significant findings were limited to the chest. The right breast and pectoral muscles were absent. There was a slightly tender radical mastectomy scar. The skin over the right upper chest showed typical marked irradiation effect. Expansion was limited on the right side. On percussion there was dullness over the entire right chest anteriorly and posteriorly. Breath sounds were decreased over the right hemithorax, and tubular breath sounds were heard over the right upper lobe anteriorly and laterally. The left lung was nomal to percussion and auscultation. The left border of the heart on percussion coincided with the left sternal border, and the heart sounds were heard over the right hemithorax. There was no evidence of recurrence of the breast carcinoma. All laboratory studies were within normal limits.

Radiographs of the chest (Fig. 6) and bronchograms showed a scoliotic deformity of the dorsal spine with the convexity to the right. There was marked deviation of the trachea

and of the cardiovascular shadow to the right, and the right lung field showed a fine linear streaking. The left lung field was normal. Bronchographic visualization of only one segment was possible on the right side, and this showed a marked saccular bronchiectasis. It appeared to be the bronchus to the anterior basal segment of the lower lobe. The bronchogram of the left lung was normal.

Because of the severity of the symptoms a right pneumonectomy was done on Oct. 5, 1950. The lung was bound to the parietal pleura by extensive, but not too dense, adhesions. The patient's postoperative course was smooth until the ninth postoperative day when she developed fever spiking to 39° C. Numerous thoracenteses were done, but pleural fluid cultures remained negative. After antibiotics were discontinued the temperature subsided. Her cough was very much improved following operation, although a slight cough remained. During her postoperative course she had numerous episodes of nausea and vomiting for which no organic basis could be found.

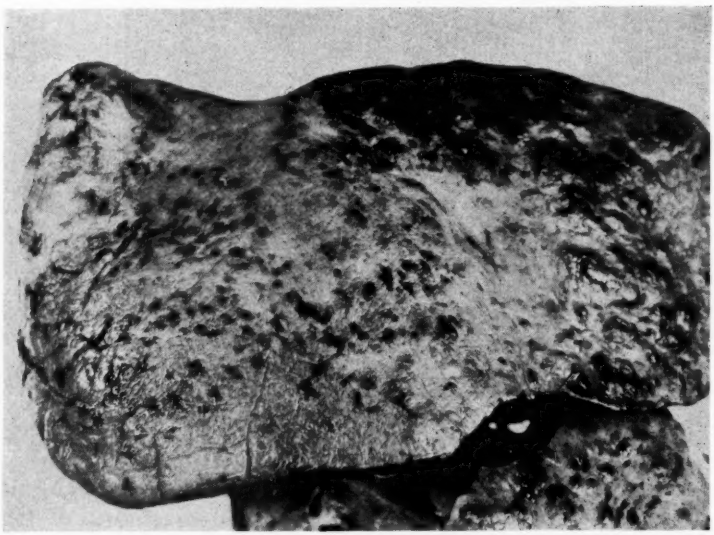

Fig. 7.—Case 2. Photograph of the upper and middle lobes of the right lung after fixation. The pleural surface is nodular and thickened, especially in the anterior portions of the lobes.

Gross description of surgically removed specimen (Surgical Pathology No. 50-4508) (Fig. 7): The specimen consisted of a small right lung which measures 14 by 11 by 4.5 cm. The lung was of firmer consistency than normal, and especially in the anterior portions of the lung crepitation was somewhat reduced, apparently without regard to segmental divisions. On the anterior aspect of all three lobes the pleura was slightly thickened and dark bluish gray. The surface was somewhat irregular and nodular. On section through the lung there was a marked difference in the appearance of the anterior and the posterior portions. Anteriorly all lobes, but especially the upper and middle lobes, were firm and gray on their cut surface. Posteriorly all lobes were essentially normal in appearance. In the anterior portion of the upper lobe and in the middle lobe mild bronchial dilatation was seen. The process is most marked in the anterior basal segment of the lower lobe where small saccular dilatations were found.

Microscopic description: Sections from various areas of the lung showed a reaction which, although qualitatively similar in all parts, varied in intensity in different pulmonary segments. The most severely affected portions were the pectoral segment of the upper lobe and the middle lobe. The anterior basal segment and the apical segment of the lower lobe also showed a marked although somewhat milder reaction. Definite relatively early changes were apparent in the apical segment of the upper lobe and in the middle basal segment of

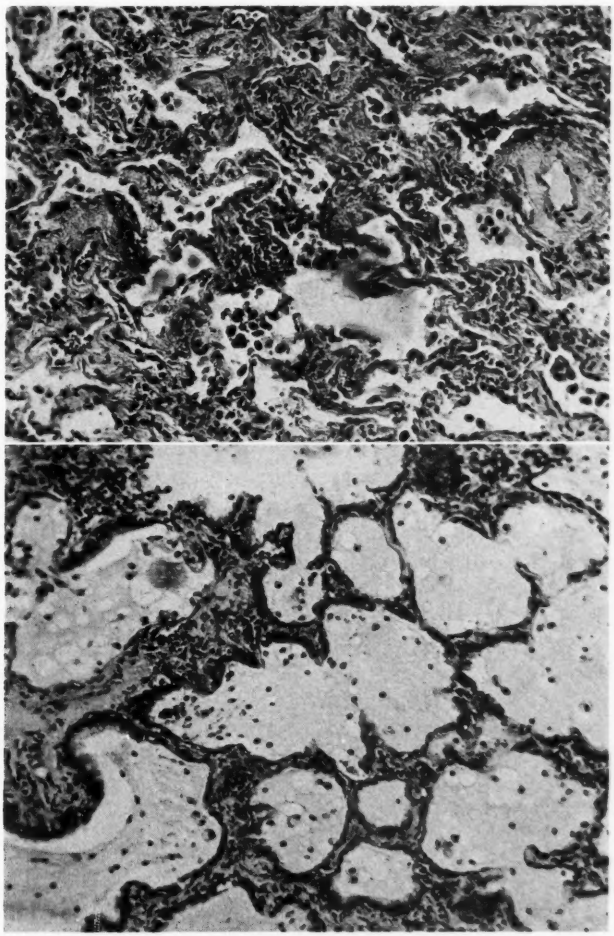

Fig. 8.

Fig. 9.

Fig. 8.—Case 2. Photomicrograph to demonstrate thickening of the alveolar walls and the presence of macrophages in the alveoli. (Hematoxylin and eosin stain ×200.)

Fig. 9.—Case 2. Photomicrograph of an area showing change of the alveolar lining cells to a low cuboidal epithelium. (Hematoxylin and eosin stain ×200.)

the lower lobe. The posterior basal segment of the lower lobe was essentially normal. In the least affected portions of the lung the bronchi showed moderate edema and increased number of goblet cells in their epithelium. The smaller bronchi and bronchioles were mildly dilated. The most pronounced changes occurred in the alveoli in the least affected segments. Their walls were mildly thickened, and with the Verhoeff van Gieson stain increase in the

number and size of the elastic fibers was obvious. The alveolar epithelium of many scattered alveoli was low cuboidal and quite prominent. In the more severely damaged areas the alveoli contained blood and edema fluid and many alveolar macrophages. The thickening of their walls was pronounced, and the lining cells were quite tall (Figs. 8, 9, and 10). The increase in elastic tissue was striking (Fig. 11). Bronchial changes in such areas included definite although mild damage to the bronchial cartilages of the smaller bronchi. Larger

Fig. 10.

Fig. 11.

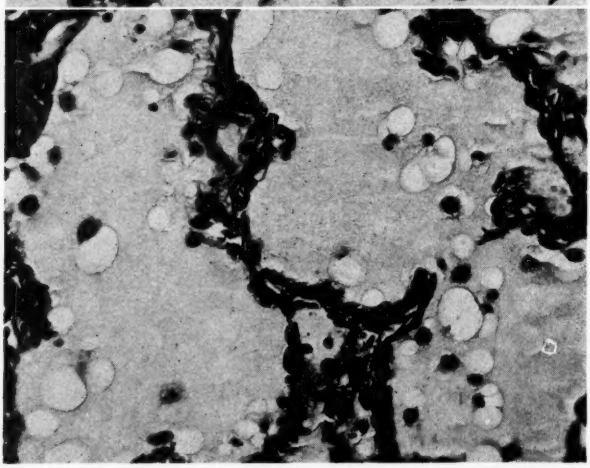

Fig. 10.—Case 2. Photomicrograph of pulmonary alveoli with localized epithelial hyperplasia. (Hematoxylin and eosin stain ×360.)

Fig. 11.—Case 2. Photomicrograph of alveolar walls with many thick elastic fibers. (Verhoeff Van Gieson stain ×400.)

bronchi showed only a mild edema. The severely affected areas of the anterior portions of the upper lobe and the middle lobe showed complete obliteration of the normal architecture by fibrosis in which many thick elastic fibers were present. No normal alveolar structures remained. Dilated bronchi were still visible, and the vessels in these areas showed edema and subintimal thickening. The pleura was edematous.

COMMENT

Both of these patients presented the typical clinical picture of irradiation damage of one lung. Cough and dyspnea were their chief complaints and were quite disabling to the patients. The radiographs of both patients were compatible with the diagnosis of radiation damage. In both of these cases pneumonectomy afforded marked relief, even though unfortunately the first case was marred by the occurrence of a postoperative empyema an unusually long time after operation. The question arises whether this complication may not in some way be related to the effects of the irradiation which this patient received. However, histologic examination of the bronchus at the line of division yielded no valid basis for such an assumption.

The pathologic findings in the two lungs are of special interest because these two cases represent the first human lungs with irradiation damage studied after surgical removal instead of post-mortem. The main features of the reaction as found in experimental animals and in autopsied human cases were seen in these cases. However, in both cases hyaline membranes, the finding regarded as so constant in autopsy material by Warren and his co-workers were absent. If one recalls that such membranes likewise were not seen in experimental animals, it becomes probable that this phenomenon to which much diagnostic significance has been attributed in the past is a terminal or agonal artefact. This view is further strengthened by Farber and Wilson's work,[16] who were able to produce such membranes artifically by a variety of means even after death in experimental animals. The alveolar epithelial hyperplasia found in Case 2 is strikingly similar to that described in rabbits by Lüdin and Werthmann.[6] The absence of such changes in Case 1 probably is due to the more advanced, fibrotic condition of this lung. In the surgically removed lungs, as well as in an autopsy case which we recently reviewed, the elastic tissue increase was marked. Such changes occur only in few other conditions, such as in healed pulmonary infarcts and apical scars,[19] and are not seen in lungs with thickened alveolar walls from other causes. In a surgically removed pulmonary lobe which had been exposed to very mild radiation, a similar marked increase in elastic tissue was seen, even though otherwise the specimen showed virtually none of the stigmata of irradiation damage. We believe that this feature of the irradiation reaction deserves emphasis as a valuable criterion for histologic diagnosis.

At the present time we believe that pneumonectomy should be employed in the treatment of unilateral irradiation damage in those cases where the severity of the patient's symptoms warrant such a procedure. Whether the original tumor for which the irradiation was administered is cured or only temporarily arrested is a secondary consideration, provided that the clinical history and examination make it appear that the patient will probably survive for a reasonable length of time. No hard and fast rules can or should be laid down in this respect. Even a patient whose ultimate prognosis is in doubt should be given the benefit of palliation of distressing respiratory symptoms which can be achieved by pneumonectomy. Undoubtedly many patients who receive x-ray

therapy to the chest sustain milder degrees of pulmonary damage. When such a condition goes unrecognized, an erroneous diagnosis of pulmonary metastatic carcinoma may lead to the administration of further x-ray therapy. Thus a relatively innocuous and largely transient irradiation reaction may be converted into an incapacitating and frequently fatal disease. Because of this hazard we urge more general awareness of the pulmonary irradiation syndrome and greater caution in applying irradiation to the thorax.

SUMMARY AND CONCLUSIONS

1. Exposure of human and animal lungs to ionizing irradiation produces a reaction whose pathologic appearance has been studied extensively.

2. In man the structural alterations resulting from exposure to relatively large quantities of irradiation cause symptoms of cough, dyspnea, chest pain and hemoptysis. Pulmonary function is markedly compromised by the process.

3. While there is no known non-operative treatment of irradiation damage of the lung, pneumonectomy in a unilateral case is an effective therapeutic measure. Two patients with this disease who were operated on with palliation of symptoms are reported.

4. Study of surgically removed lungs showed that one of the criteria for the histologic diagnosis of irradiation damage in autopsy material, the finding of hyaline membranes, is probably an artefact not seen in surgically removed lungs. The presence of increased number of thick elastic fibers diffusely throughout the irradiated lung appears to be an important criterion for the histologic diagnosis of irradiation damage.

REFERENCES

1. Groover, T. A., Christie, A. C., and Merritt, E. A.: Observations on the Use of the Copper Filter in the Roentgen Treatment of Deep-seated Malignancies, South. M. J. **15**: 440-444, 1922.
2. Graham, E. A., and Singer, J. J.: Successful Removal of an Entire Lung for Carcinoma of the Bronchus, J. A. M. A. **101**: 1371-1374, 1933.
3. Desjardins, A. U.: The Action of Roentgen Rays and Radium on the Heart and Lungs, Am. J. Roentgenol. **28**: 271-292, 421-436, 567-578, 701-720, 845-858, 1932.
4. Engelstad, R. B.: Über die Wirkungen der Röntgenstrahlen auf die Lungen. Acta. radiol., supp. **19**: 1-94, 1934.
5. Warren, S.: Effect of Radiation on Normal Tissues. V. Effects on the Respiratory System, Arch. Path. **34**: 917-931, 1942.
6. Lüdin, M., and Werthemann, A.: Lungenveränderungen nach experimenteller Röntgenbestrahlung, Strahlentherapie **38**: 684-701, 1930.
7. Warren, S., and Gates, O.: Radiation Pneumonitis: Experimental and Pathologic Observations, Arch. Path. **30**: 440-460, **1940.**
8. Engelstad, R. B.: Pulmonary Lesions After Roentgen and Radium Irradiation, Am. J. Roentgenol. **43**: 676-681, 1940.
9. Warren, S., and Spencer, J.: Radiation Reaction in the Lung, Am. J. Roentgenol. **43**: 682-701, 1940.
10. Evans, W. A., and Leucutia, T.: Intrathoracic Changes Induced by Heavy Radiation, Am. J. Roentgenol. **13**: 203-220, 1925.
11. Freid, J. R., and Goldbert, H.: Post-irradiation Changes in the Lungs and Thorax, Am. J. Roentgenol. **43**: 877-895, 1940.
12. Landau, W.: Lungeninduration Infolge Röntgenbestrahlung des Brustkorbs bei Mammakarzinom, Ztschr. f. Tuberkulose **65**: 212-219, 1932.
13. Belt, T. H.: Über Tödliche Lungenfibrose bei Gewerblicher Radiumschädigung, Pathologisch-anatomischer Teil, Frankf. Ztschr. f. Path. **42**: 170-187, 1931.

14. Tönges, E., and Kalbfleisch, H. H.: Ein Zweiter Fall von Tödlicher Lungenfibrose Infolge Gewerblicher Radiumeinwirkung, Frankf. Ztschr. f. Path. **50**: 100-122, 1936.
15. Schairer, E., and Krombach, E.: Röntgenschädigung der Lunge mit Tödlichem Ausgang, Strahlentherapie **64**: 267-290, 1939.
16. Farber, S., and Wilson, J. L.: The Hyaline Membrane in the Lungs, Arch. Path. **14**: 437-460, 1932.
17. Leach, J. E.: Abnormal Pulmonary Physiology as a Result of Chronic Irradiation Pleuropneumonitis, Am. J. Roentgenol. **50**: 772-778, 1943.
18. Baldwin, E. deF., Cournand, A., and Richards, D. W.: Pulmonary Insufficiency. II. A Study of Thirty-nine Cases of Pulmonary Fibrosis, Medicine **28**: 1-25, 1949.
19. Castleman, B.: Healed Pulmonary Infarcts, Arch. Path. **30**: 130-142, 1940.

DISCUSSION

DR. EVARTS A. GRAHAM, St. Louis.—Our reason for bringing this subject before you this morning is not because there is anything new or dramatic about it, but rather to make us all more or less aware of what this very serious condition is. It has been well described before, as Dr. Bergmann indicated; it has been produced experimentally by many workers; there is no difficulty about that in experimental animals. The main thing is, however, that if none of you has seen a patient suffering from this condition you cannot have any idea of how miserable such a person is. I think anybody would much rather be dead than he would be to have this condition.

These two women whom we saw were unable to sleep. Walking even a short distance such as two or three times across the room set up violent dyspnea, terrific harassing cough. One woman had become accustomed to taking large amounts of narcotics because that was the only way she could get relief. Both women said voluntarily that if they had had any idea that their operations for cancer were to be followed by such a condition as this—they thought it was partly due to the operation rather than x-ray treatment—they would far rather be dead from the cancer than to suffer as they were suffering.

Every rose has its thorns, and the thorns of the rose of x-ray therapy applied to the lung is this condition. You are looking, of course, at extreme examples. Dr. Bergmann mentioned in his presentation that there are all gradations, and I am sure that probably there is nobody in this room who has not seen a patient, maybe not having recognized it as such, who had a bad cough following irradiation therapy over the lung field. The explanation of the cough is not apparent right now, because there does not seem to be any ulceration of the bronchial tree, but probably it is due to irritation of the nerve endings. At any rate, whatever the explanation may be, it is a most terrible condition.

All people, as indicated by Dr. Bergmann in the paper, and as has been brought out before, are not equally susceptible to irradiation effects. Fortunately, this apparently occurs in only a small percentage of people who get the usual therapeutic amount of x-ray therapy. But it does occur, we do not know in just how large a percentage. Unfortunately also, in the case of these two patients, we have been unable to find out with any degree of accuracy what the dosage was, because they were irradiated in a different institution.

DR. HERBERT D. ADAMS, Boston.—This problem has interested us a great deal because we have had a large number of cases in which we have used x-ray therapy not only in the treatment of primary lesions within the lung but, in addition, for treatment of adjacent lesions which entailed considerable simultaneous irradiation of the lung. The most common of the latter, of course, are the cases of carcinoma of the breast.

We have found, just as Dr. Graham has said, that we must consider the possibility of very serious pulmonary damage, particularly if too much x-ray is given to this region. It has been our experience also that patients with severe irradiation damage to the lung are thoroughly incapacitated and miserable from intractable cough and dyspnea. Only pneumonectomy can relieve these patients, but I wish to call your attention to the importance of careful pulmonary function tests before considering pneumonectomy in this condition. It is impossible otherwise to tell whether there has been some damage to the function of the opposite lung or to estimate accurately whether there is adequate function of the opposite lung to maintain the patient after pneumonectomy. We have had two cases in the past year which

illustrate the value and importance of carrying out complete pulmonary function tests in these patients. Both these patients had radical mastectomies and x-ray therapy elsewhere in 1947. The first patient, a women 65 years of age, had very severe damage of the left lung. A bronchoscopy and bronchogram were done to rule out bronchial stenosis and, as can be seen from the bronchogram, no great degree of bronchiectasis or obstruction was demonstrated. In this particular instance we were able to find out fairly accurately what the dosage was, and the patient received four courses of x-ray over a period of several years, amounting to the tremendous dosage of over 17,000 r. The maximum breathing capacity in this patient was down to 58 per cent of that predicted. Differential bronchospirometry showed that her badly damaged lung, however, was still carrying 26 per cent of the remaining function. We thought, therefore, that we could not possibly remove the lung safely and this patient, as Dr. Graham has pointed out, was so miserable that she was extremely disappointed that we could not offer her surgery.

The other patient, a woman 35 years of age, had marked retraction and fibrosis of the right lung, and the bronchogram shows a greater degree of bronchiectasis than in the first case. In addition, the good lung is slightly overdistended and emphysematous. In this instance we were not able to get the exact dosage, although the report we received was that she had a single course standard dosage of roughly 6,000 to 8,000 r. This patient's maximum breathing capacity was 93 per cent of that predicted, and bronchospirometry showed that the damaged lung was carrying only 10 per cent of that function. We were able, therefore, to carry out pneumonectomy, and she did very well.

Our roentgenologists have thought that perhaps, in spite of the fact that most of these patients have received a greater dosage than they should have received and this was probably the etiology in these cases, in some instances standard dosage has also produced severe irradiation pneumonitis. It is their opinion that perhaps an associated infection has been the primary factor, or at least an important contributing factor, in some of these more severely damaged lungs. As a result they have been carrying on antibiotic treatment throughout the period of x-ray therapy and during the posttherapy period of maximum reaction. Further evidence that this may be an important factor is the fact that in the case that we considered inoperable (Case 1), this patient has improved a great deal on a medical regimen of prolonged antibiotic therapy.

In addition, in such cases as carcinoma of the breast, carcinoma of the esophagus or any other adjacent malignancy where the lung may become irradiated in the course of x-ray therapy, they have been resorting more and more to high voltage two-million volt x-ray in an additional effort to try to avoid this type of lung damage.

I would appreciate very much Dr. Graham's or Dr. Bergmann's opinion regarding these etiologic factors and these specific measures in possibly preventing this serious pulmonary damage.

DR. ADRIAN LAMBERT, JR., New York.—In line with Dr. Adams' remarks about pulmonary function, I should like to ask whether in these cases they develop a pulmonary hypertension and whether the pulmonary arteries develop elastic fibers that show the same changes as in the lung tissue.

DR. OTTO C. BRANTIGAN, Baltimore.—Radiation fibrosis of the lung can be produced experimentally in animals, and it has also been shown that it can be prevented by anticoagulant therapy with heparin. Dr. Stanley Macht, formerly our radiologist at Baltimore City Hospitals, and myself, wondered if radiation fibrosis of the lung could be prevented in the human being with anticoagulant therapy. We tried it on six or eight patients with inoperable carcinoma of the lung or carcinoma of the esophagus. During radiation therapy adequate anticoagulant therapy was carried out, using dicumarol. None of the patients lived longer than three months after radiation therapy. We did obtain an autopsy on these patients and none showed radiation fibrosis of the lung, either grossly or microscopically. The patients all died of distant metastasis, even though the primary carcinoma receded or almost completely disappeared.

DR. GEORGE W. WRIGHT, Saranac Lake.—These two cases are of considerable interest to those who are working actively in the field of physiology. It has long been suspected on the basis of pretty good evidence that there are stimuli arising within the lung and chest wall that may drive the respiratory center in an abnormal manner. The demonstration by these two cases of the fact that overbreathing was markedly reduced after resection, seems to me to quite convincingly prove that there are stimuli arising in the lung that do drive the respiratory centers. This same situation has its counterpart in a number of nonsurgical diseases; for example, it is beautifully demonstrated in the pulmonary granulomatosis of beryllium workers; also demonstrated in some cases of Boeck's sarcoid. These diseased individuals show very definite overbreathing, and in the case of pulmonary granulomatosis, the overbreathing is far out of proportion to the hypoxia and persists in spite of raising blood oxygen levels by breathing pure oxygen. These cases respond very beautifully to cortisone for the period of time they are under treatment. I am wondering whether or not that might be tried in cases of pulmonary damage secondary to irradiation.

The other clinical entity accompanied by overbreathing is that of the thickened pleura so commonly seen after pneumothorax. Overbreathing is very common in these and decortication produces a reduction in the degree of overbreathing. I am intrigued also with the possibility of nerve section in treating these patients who overbreathe because of a stiff or relatively nonexpandable lung or chest wall.

DR. BERGMANN (closing).—I want to thank all the discussers for their valuable suggestions. Dr. Adams' cases were especially interesting because we were not able to do as complete pulmonary function studies as he was able to do. In the first case such studies were tried, but the patient had a procaine reaction while we were attempting to do bronchospirometry and almost died, and so the procedure was abandoned. In the second case there was no question of pulmonary insufficiency of the other lung, and physiologic studies were not carried out, although we did do bronchoscopy and, of course, bronchograms on both patients.

Dr. Lambert asked about pulmonary artery changes. We have not been able to demonstrate any changes in the pulmonary arteries, and of course it is well known that blood vessels are relatively resistant to the influence of ionizing irradiation. We are familiar with the published work on heparin prophylaxis of irradiation damage of the lung. It sounds very interesting, and I think it certainly should be investigated further.

As for Dr. Wright's remarks, I think cortisone might be worth a trial in these cases, since one of the important aspects of this lesion is an increased amount of fibrous tissue. Since it has been shown in many other conditions that cortisone and ACTH tend to reduce fibroplasia, these drugs might be very helpful in pulmonary irradiation damage as well.

I doubt that decortication would be possible in these patients because there does not seem to be a peel over the pleura. The pleura is thickened and edematous, and I do not believe that one could find a line of cleavage for decortication of irradiated lungs.

RECONSTRUCTIVE SURGERY OF THE TRACHEA AND BRONCHI: LATE RESULTS WITH DERMAL GRAFTS

PAUL W. GEBAUER, M.D.
HONOLULU, T.H.

IDEAL surgery directly treats the disorder, and preserves a physiologic state as nearly normal as possible. The surgical treatment of pulmonary disease is emerging from resection at the lung and lobe level to resection at the segmental level. Even wedge and subsegmental resections are being applied with increasing frequency. A logical consequence of this progress is the development of reconstructive surgery of the trachea and bronchi.

Segmental resection, of inestimable value in the treatment of bronchiectasis, deserves a similar status in the treatment of some tuberculous and other localized lesions of the lungs. However, the presence of central occlusive lesions of the bronchi sometimes contradict its use, as well as more localized excisions of isolated lung lesions. Similarly, a lesion of a main bronchus may prohibit lobectomy, and pneumonectomy must be done, thereby invoking a marked physiologic loss for the patient, because a direct treatment of the bronchus is not available. Also, in the absence of parenchymal lung lesions, there are instances of localized new growths, and cicatricial deformities of the bronchi, which produce symptoms, and prevent normal function of a lobe or lung. Such lesions beg for corrective bronchial surgery rather than pulmonary resection. In addition to these conditions, the realization that benign and even malignant tracheal tumors may asphyxiate their hosts before they reach a nonresectable stage is ample evidence of the need for reconstructive surgery of the air passages. This is practically a virgin field in clinical surgery, despite the sporadic endeavors of the past, which were usually directed to the trachea, primarily for asphyxia prevention instead of the conservation of good lung tissue.

Experimental tracheobronchial surgery on dogs is just now reaching a significant scope and volume. It has definitely demonstrated the feasibility of such surgery, as well as the worthy receptiveness of the tracheobronchial tree to reconstruction. It has not yet supplied a method of replacing portions of the air passages with a high degree of safety and success over a period of years. However it has again demonstrated the stubbornness and contracture of fibrous connective tissue and, therefore, the need for either viable grafts or practically permanent prostheses, or artificial support. The usefulness of plastic reconstruction without grafting or prostheses is, perhaps the most laudable contribution of the laboratory to this immature surgical field.

The experiences of the writer, partially presented in Tables I, II and III, because of their confinement to a rather small and constant population, are a fair indication of the need for reconstructive procedures on the trachea and

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bronchi. Of greater significance is the fact that two clinical reconstructive methods have been used with sufficient success to warrant the following prediction: tracheobronchial surgery, consisting of inlay and patch grafts, sleeve resection and anastomoses, tubular replacements, excisions with end-to-side anastomoses, and so on, sometime will be employed frequently in the treatment of a variety of pulmonary and air passage disorders.

Because a promising beginning has been made, it seems worth while to further the fulfillment of this prediction with a theoretical appraisal of surgical methods available, and those used experimentally, in the light of clinical conditions likely to demand tracheobronchial reconstruction. The following personal opinions and theoretical statements are concerned with the bronchi and the thoracic portion of the trachea, inasmuch as reconstruction of the cervical trachea is satisfactorily managed with surgical methods long in use.

METHODS

I. *Excision and Reconstruction Without Grafts or Prostheses.*—

This method, experimentally used by Jackson and associates,[1] is relatively simple, and undoubtedly the best now available. It can be used in a variety of ways, such as end-to-end and end-to-side anastomoses. Small wedge resections of the lateral walls of the tracheal bifurcation afford a convenient means of increasing the orifice of either main bronchus. Clinically, however, the method is not quite as applicable as might be inferred from experimental tracheal excision in dogs. In these animals, the relatively huge tracheobronchial cross section lessens the significance of excess, or constricting, scar at suture lines. Also, the excessive mobility, resulting from its considerable length, permits reapproximation without undue tension. When this is possible, there is no need for an indwelling prosthesis.

Aside from the greater technical difficulties in man, the only detriment to wide use of this method is the scarcity of suitable lesions. In general, these are localized chronic inflammations, fistulas, benign tumors, and short, fibrous strictures resulting from trauma or infection. Very early, small malignant tumors, conceivably, could be removed with this method, but such treatment would violate the present-day principles of cancer surgery. Healed tuberculous strictures occasionally involve only short tracheobronchial segments. Such lesions are not infrequent, and are well suited to this method. Furthermore, its use may be simplified by the necessity of some type of pulmonary resection.

Tuberculous disease of the upper lobe bronchi frequently extends to the immediately proximal main bronchi, and stenoses of both structures may occur with healing. More often than not, removal of the upper lobe is indicated because of disease or changes within it consequent to its bronchial disease. If the main bronchial lesion is not too long, it can be excised with the upper lobe, and the bronchial stump of the lower lobe sutured to the distal trachea. Tension on the anastomosis is greatest when the lung is fully expanded, hence it is facilitated by removal of some of the lung, for the flare of the pulmonary hilus is reduced so that it can be placed more deeply into the mediastinum, without excess strain.

TABLE I. BRONCHIAL DERMAL GRAFTS

CASE	DISEASE	OPERATION	TIME POST-OPERATIVE	SPUTUM		REMARKS	PRESENT STATUS
				PRE-OPERATIVE	NOW		
3	Atelectasis RUL. Severe stenosis right main bronchus. Active tuberculous upper lobe bronchus.	Right upper lobectomy. Dermal graft right main bronchus.	2 yrs., 7 mo.	Pos.	Neg.	Graft wire removed 16 mo. p.o. Mild recurrent tuberclosis lower trachea 19 mo. p.o. healed with PAS and SM	Home. Working. Small but adequate bronchial lumen.
4	Atelectasis LUL. Stenosis left main bronchus. Right pneumothorax.	Left upper lobectomy. Dermal graft left main bronchus.	2 yrs., 7 mo.	Pos.	Neg.	Left one-stage thoracoplasty done 8 mo. p.o. for nodular infiltration in expanded lower lobe apex.	Home. Working. Good bronchial lumen.
5	Emphysema. Incomplete thoracoplasty left. Stenosis distal left main bronchus.	Left upper lobectomy. Dermal graft left main bronchus.	2 yrs., 6 mo.	Pos.	Neg.	Tuberclosis cured by lobectomy. Respiratory status improved by better aeration LL lobe.	Home. Light work. Good bronchial lumen.
6	Atelectasis LUL. Severe stenosis left main bronchus.	Left upper lobectomy. Dermal graft left main bronchus.	2 yrs., 6 mo.	Pos.	Neg.	Operative trauma to left recurrent laryngeal nerve caused left vocal cord paresis which recovered 18 mo. p.o.	Home. Working. Excellent bronchial lumen.
7	Atelectasis RUL. Severe and very thick stenosis right main bronchus.	Right upper lobectomy. Wide diamond-shaped dermal graft RMB.	2 yrs., 4 Mo.	Neg.	Neg.	Early partial detachment of too wide a graft. No leakage. P. O. dilatations effective.	Home. Working. Good bronchial lumen.
8	Atelectasis RUL. Severe stenosis right main bronchus.	Right upper lobectomy. Dermal graft RMB.	2 yrs., 3 mo.	Pos.	Neg.	Recurrent tuberclosis lower trachea, and proximal RMB. Wire removed 1 year p.o. Good healing with long course of PAS and SM.	Ambulant ward. No. bronchial symptoms. Soon ready for discharge.
10	Borderline respiratory status. Destruction LUL. Healed bilateral tuberculous bronchitis. Stenosis LMB.	Left upper lobectomy. Dermal graft distal LMB. Modified thoracoplasty.	2 yrs.,	Pos.	Pos.	Bronchial lumen is rather small and there is some bronchiectasis in the residual LLL. Respiratory status improved.	General condition excellent. Source of positive suptum uncertain.
11	Considerable nodular, arrested infiltration left lung. Severe stenosis LMB.	No resection. Dermal graft left main bronchus.	1 yr., 11 mo.	? Pos.	Neg.	Good graft result. Resection of left lingula probably should have been done.	Home. Not working. No bronchial symptoms
12	Severe stenosis LMB. Tuberculosis and bronchiectasis left upper lobe.	Left upper lobectomy. Dermal graft LMB.	1 yr., 10 mo.	Pos.	Neg.	Excellent graft and result.	Home. Working. No symptoms.
14	Atelectasis RUL. Severe stenosis RMB.	Right upper lobectomy. Dermal graft RMB.	10 mo.	Pos.	Neg.	Complete symptomatic relief.	Home. Not working. Excellent bronchial lumen.

TABLE II. TRACHEAL GRAFTS AND TRACHEAL PLUS BRONCHIAL GRAFTS

CASE	DISEASE	OPERATION	TIME POST-OPERATIVE	SPUTUM PRE-OPERATIVE	SPUTUM NOW	REMARKS	PRESENT STATUS
9	Severe stenosis trachea and right lower lobe bronchus. Right basal segmental atelectasis.	Dermal graft of trachea and second dermal graft of right bronchus. No resection.	2 yrs., 2 mo.	Neg.	Neg.	Right pneumonectomy done for bronchiectasis 1 year p.o. Excellent, healed viable graft.	In hospital ambulant ward. Recovering from GI disease. Good tracheal lumen.
13	Severe stenosis trachea and RMB. Minimal infiltration right lung. Atelectasis middle and lower lobes immediately prior to surgery.	Dermal graft trachea. Right pneumonectomy.	–	Pos.	–	Died third p.o. day—asphyxia. Obstructive edema of ungrafted upper trachea and left main bronchial orifice.	Dead
15	Severe stenosis trachea, left main bronchus, and right intermediate bronchus. Atelectasis RML and RLL. Minimal infiltration RUL.	1. Tracheotomy. 2. Dermal graft LMB. 3. Dermal graft trachea and right middle and lower lobectomy.	4 mo.	Pos.	Neg.	Considering patient's preoperative asphyxial state present status is unbelievable. A miraculous result.	Good condition. Perfect LMB graft. Adequate tracheal lumen. Tracheotomy tube out 10 weeks.

TABLE III. OTHER TRACHEOBRONCHIAL CASES

CASE	DISEASE	OPERATION	TIME POST-OPERATIVE	SPUTUM PRE-OPERATIVE	SPUTUM NOW	REMARKS	PRESENT STATUS
1	Severe stenosis whole trachea and proximal RMB	Attempted excision of trachea and replacement with wire coil and fascia.	–	Pos.	–	Died at operation before reconstruction could be initiated.	Dead
2	Severe stenosis distal half of trachea and RMB. Destruction of right lung.	Attempted right pneumonectomy and tracheal graft.	–	Pos.	–	Died at operation before reconstruction could be initiated.	Dead
16	Severe stenosis right main and upper lobe bronchi. Tuberculous middle lobe. Old right thoracoplasty.	Right upper and middle lobectomy. Excision of RMB. Anastomosis right lower lobe bronchus to trachea.	2 mos.	Pos.	Neg.	Rapid, complete healing noted bronchoscopically.	Good condition. Convalescing. No bronchial symptoms

Case 16, illustrated in Figs. 1, 2, and 3, is an excellent example. It is probably the first instance of elective excision of a whole main bronchus. The bronchial specimen contained five fairly intact cartilages, and two others had probably been destroyed in the central area of severe stenosis. The patient had severe stenosis of the right main and upper lobe bronchi, bronchiectasis of the upper lobe (under a five-rib thoracoplasty done ten years previously), and active

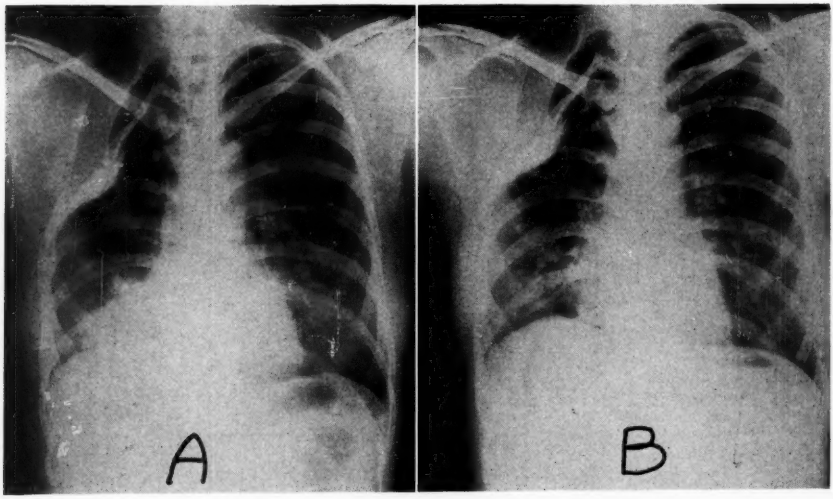

Fig. 1, *A* and *B*.—(Case 16) Preoperative roentgenograms. *A*, Eighteen months before operation, old right thoracoplasty, acute inflammation and stenosis right main bronchus at carina, and pneumonic "spread" right middle lobe. *B*, Just before operation, right main bronchus healed with severe stenosis; middle lobe not entirely clear; sputum positive.

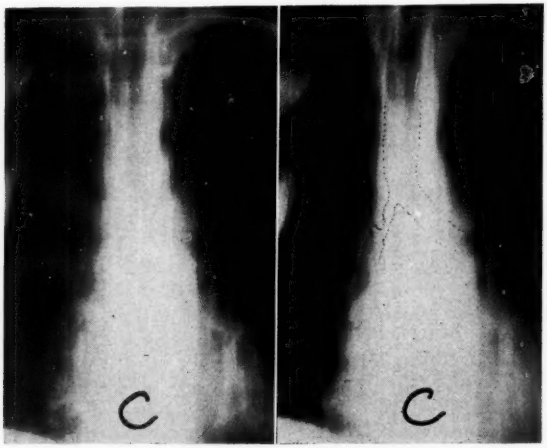

Fig. 1, *C*.—Unmarked and marked preoperative planigrams show severe stenosis of right upper lobe and right main bronchi.

tuberculosis of the middle lobe. The upper and middle lobes were removed. The right bronchus was excised from the carina to the middle lobe stump. Because of the absence of the right middle and upper lobes, no difficulty was encountered displacing the right lower lobe bronchus upward about 2 inches, and anastomosing it to the trachea. Bronchoscopy now reveals a good lumen to the right basal bronchi which are opposite the left main bronchial orifice.

On the left side, the subaortic space is small, and the flare of the hilus might cause difficulties. However, its reduction by partial pulmonary excision would probably permit substantial bronchial excisions and anastomoses.

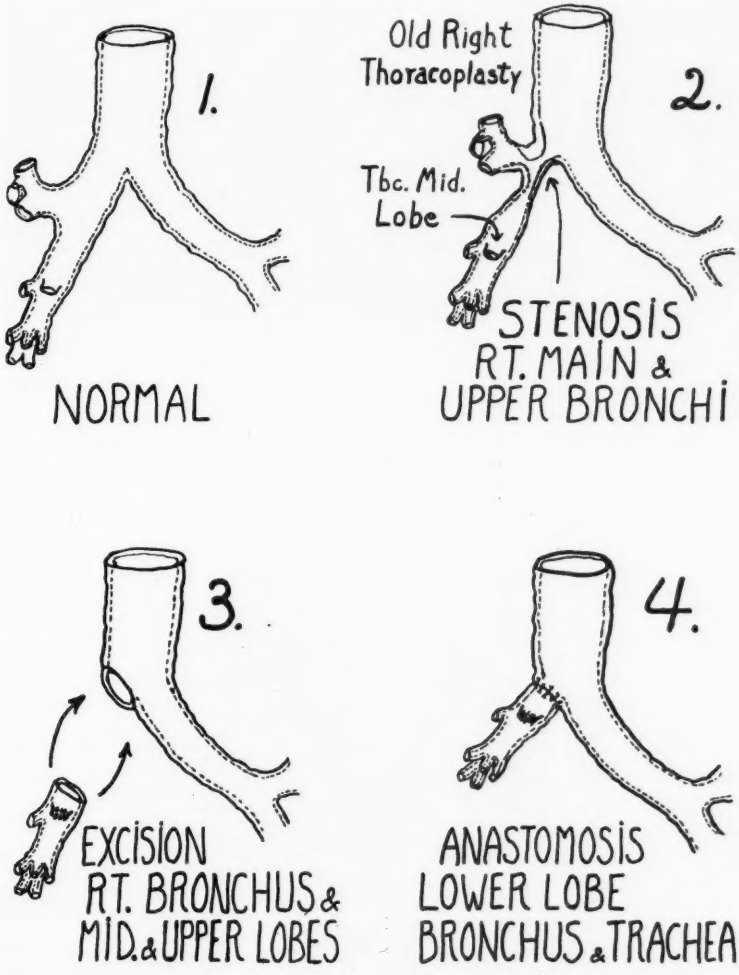

Fig. 2.—(Case 16) Diagrams illustrating the conditions encountered at operation and the treatment applied. The upper and middle lobes were removed. The lower lobe was grossly worthy of preservation, so the stenotic right bronchus was excised, and the lower lobe bronchus anastomosed to trachea.

The advantages of total division of the inferior pulmonary ligament and complete mobilization of the residual lobes are obvious. These procedures are usually indicated in all types of partial pulmonary excisions. An open type of

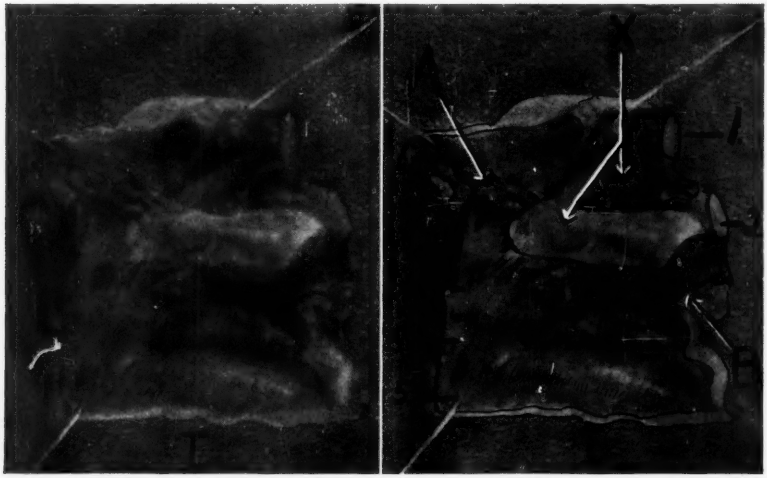

Fig. 3, *I.*—(Case 16) Posterior view of bronchial specimen. *I*, Right bronchus which has been formalin fixed, and is therefore shrunken. It has been dissected, and much scar removed at *X*, so that it could be opened out. *A*, Tracheal side of the main bronchial stenosis; *B*, upper lobe stenosis; *1* to *5*, partially intact bronchial cartilages, apparently two others were destroyed and replaced by cicatrix.

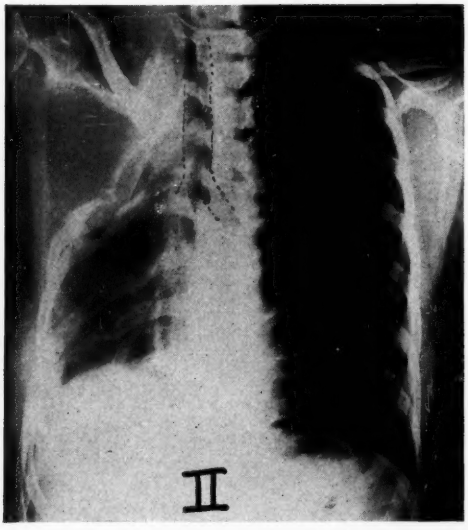

Fig. 3, *II.*—Roentgenogram one month postoperative. Left anterior oblique roentgenogram one month after operation, the basal branches of the right lower lobe are poorly visualized. Bronchoscopically they are visible opposite the orifice of the left main bronchus.

anastomoses, absolute avoidance of any type of bronchial clamp, and a single row of meticulous, simple interrupted sutures of atraumatic, fine multistrand wire are recommended. The only criticism of the everted cuff suture is the loss of structural length which, in man, may be rather precious. The cylinder of cellular foreign body reaction about nonabsorbable sutures, other than wire, is undesirable.

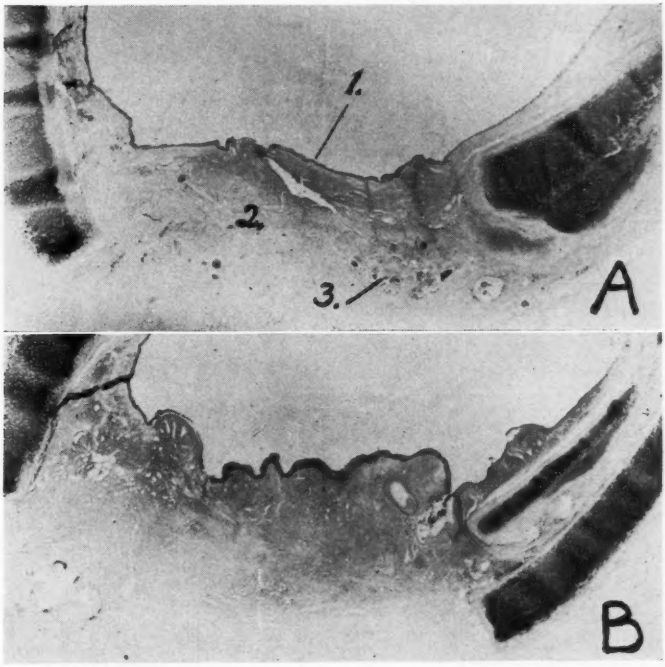

Fig. 4.—Dermal graft, dog's trachea, five weeks after insertion. *A*, Section through an area where epidermis was well removed. *1*, Bronchial epithelium completely covers the graft. *2; 3* indicates a slight residue of the generous distribution of hair follicles found in fresh dog's skin. *B*, Section near extremity of graft where epidermis was not well removed reveals thin squamous epithelium on graft.

II. *Autografts.*—

1. *Dermal skin supported with wire:* When a considerable length of the tracheobronchial tree requires repair or excision, Method I may be inadequate. This is the nature of many healed tuberculous strictures, and their correction with wire-supported dermal grafts, as previously reported,[2, 3] has proved most satisfactory, as demonstrated by Tables I, II, and III.

The available evidence indicates that these grafts are not converted to fibrous tissue, but retain viability, as well as their histologic, collagenous, and elastic characteristics. They usually remain as inserted, an ever-present example of how the job was done. They may survive recurrent tuberculous infection. Even in the dog, hair follicles rapidly disappear (Fig. 4). Some sweat glands

survive for at least a year, at which time only scattered remnants of sebaceous elements can be found. If the epidermis is not well removed it may persist, and in those areas the tracheobronchial lining may be a squamous type of epithelium. This may be without apparent physiologic effect, as it is in one tracheal graft in this series, which contains a central area of squamous epithelium. All the other grafts appear to have mucosal coverings identical to the

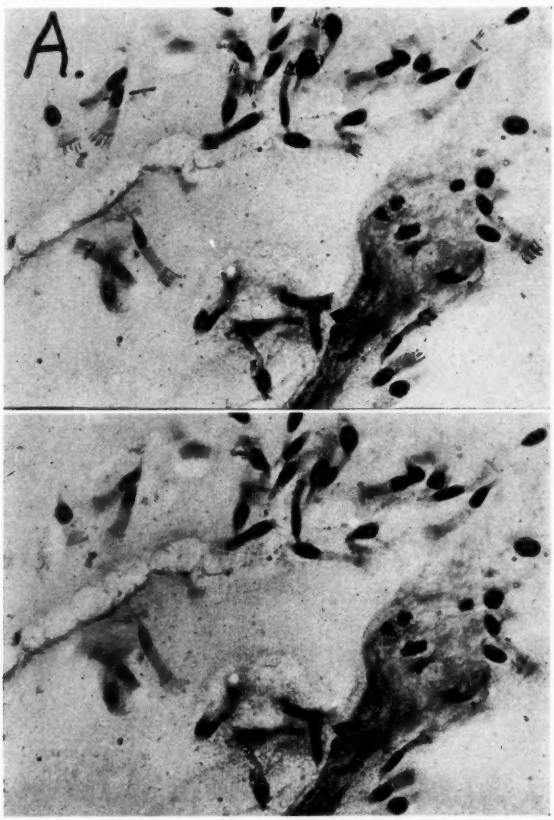

Fig. 5.—Sample of epithelium curretted from a dermal bronchial graft over two years after operation. Many normal ciliated cells are present. (Hematoxylin and eosin stain, ×530.)

neighboring bronchi. Bronchoscopic epithelial currettings have been taken in five patients from five weeks to over two years after operation. Ciliated epithelial cells have been encountered in all these specimens. Other cellular elements may be prominent up to two months; thereafter, epithelium, histologically indistinguishable from the normal respiratory type, has been encountered. A single field of a sample is illustrated in Fig. 5. The facts that dermal skin is always available in abundance and that every surviving patient has had complete, or marked, symptomatic relief are highly significant.

In fibrous strictures this procedure does not call for the removal of any portion of the trachea or bronchus, it simply adds to the circumference of either structure along the length of the stenotic area. This is gained by spreading apart the edges of a longitudinal incision through the healed stenosis. The resulting defect is held open and filled with the graft. The method should be

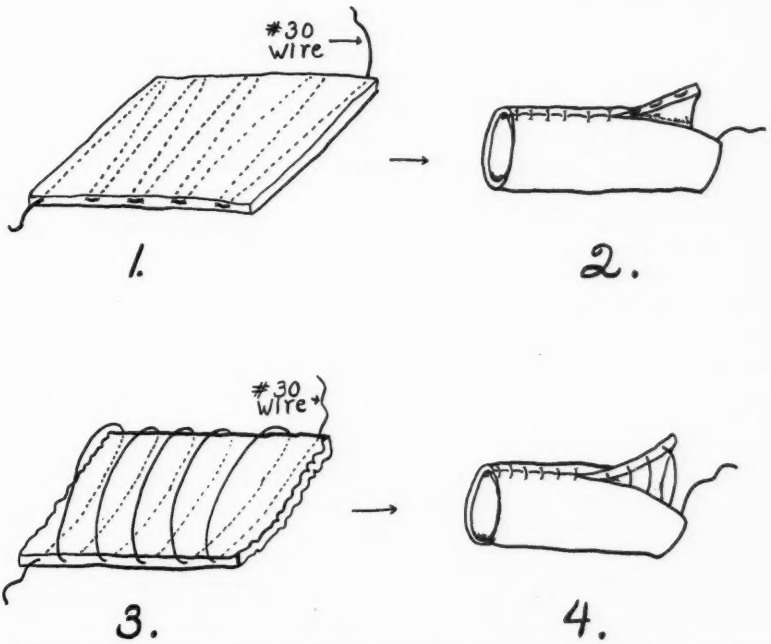

Fig. 6.—Drawings illustrating two methods of preparing wire-supported, tubular dermal grafts for experimental purposes, the dermal skin must be thick. *1*, Wire inserted from side to side as in an ordinary, elliptical, inlay graft. *2*, Graft is curled into a tube. The longitudinal closure is easier if done after the ends of the graft have been sutured to the tracheobronchial structures. With this method the graft can be slid under the tracheal tube, and the latter does not have to be removed. *3*, Wire inserted as a continuous coil with long free loops. When insertion is completed, a piece of sterile, suitable, wooden dowl is placed under the free wire loops, the wire drawn tight, the graft pushed around the dowl into approximation, and the longitudinal sutures placed, *4*. This must be done before the graft is inserted, it is a more tedious procedure, and the wire coil affords less support.

useful for the correction of tracheobronchial defects other than the post-tuberculous, such as those incident to the removal of benign lesions, and especially those of the distal trachea, arising from excision of bronchial carcinomas, which have extended into the trachea, beyond the ordinary limits of resection.

Wire-supported dermal skin has not been used in the form of a tubular graft, such as might be required following a sleeve resection, although the preparation of this type of graft is not too difficult. Two methods are illustrated in Fig. 6. Because a free graft of this much surface is contrary to well-founded surgical principles, it has not been used clinically, even though its chances of satisfactory survival are considered better than those of fascia lata.

2. *Bronchial autografts:* Some of the early postoperative experimental results reported by Daniel and associates[4] were most promising. In these grafts artificial support is not necessary, and a graft, more suitable than a patch of the patient's own tracheobronchial structure, is hard to imagine. However, in man, such tissue is supplied only in sufficient quantity by the main and lobar bronchi, and the probability of its clinical application is immediately diminished when one realizes that the usual combination of circumstances appropriate to its use are distinctly unique. It may embrace the necessity for a certain type of pulmonary resection, including uninvolved bronchial structures, in addition to a distant tracheobronchial lesion in need of excision or repair. Thus, an excised main bronchus might be used to correct or fill a tracheal defect, and possibly a normal lobar bronchus from an excised lobe could replace a diseased lobar bronchus. It is also conceivable that removal of a segment of a main bronchus followed by its anastomosis would supply a graft for a remote defect in the trachea, for example. In this manner pulmonary resection might be avoided. As with dermal grafts on rare occasions, the limits of bronchial and carinal excision might be extended, if some parts of the removed bronchus were suitable for a tracheal graft. This would demand an extraordinary prepneumonectomy appraisal and judgment. The rarity of such complex circumstances is quite apparent, contrary to the hopeful prophesies of Carter and associate,[5] who used this method experimentally, and reported postoperative results up to nine months. Despite the strong surgical appeal of autografts, they should not be regarded as applicable in most situations, for the most common disease requiring pulmonary resection in combination with tracheobronchial reconstruction is tuberculosis. In this particular situation a great majority of the excised bronchi are clearly unsuitable as grafts.[6]

Regarding technique: in dogs, the difficulties of ventilation with this type of surgery are aggravated by an open mediastinum. In man, if one pleural sac is intact, similar difficulties are more easily handled by a number of methods.[7] Generally, in tubation of the contralateral main bronchus with the intratracheal catheter permits adequate ventilation as well as anesthesia, and unhurried, open tracheobronchial procedures can be done.

The advisability of temporary postoperative drainage, preferably constant suction, is unquestioned in all instances of partial pulmonary resection and potential tracheobronchial air leaks. After tracheobronchial reconstruction, even with one pleural sac intact, it is considered an absolute necessity. In the twenty experiments reported by Carter and Strieder,[5] at least six of eighteen dogs died, evidently from lung collapse probably due to air leaks. It is inconceivable that postoperative suction, drainage, or aspiration were not used. On the other hand it is equally inconceivable that all these early deaths would have occurred if these necessary procedures had been employed. In man, an airtight graft at operation is not insurance against subsequent leakage, especially in the first twenty-four hours. With adequate suction and drainage, such leakage is without consequence and ceases spontaneously. In the experience of the writer, every patient has been on constant suction for a minimum of forty-eight hours, even though significant temporary leakage occurred only twice.

A patch graft inserted as an onlay within the tracheobronchial lumen is forced into its bed by the high pressures of forced expiration, thereby lessening the tendency to air leakage. In the relatively large tracheobronchial structures of dogs such intraluminal grafts may have little significance. However, a patch graft within the bronchial lumen of a small woman could easily be obstructive, and it certainly would be a distinct subtraction from the lumen gained by a reconstructive procedure. Theoretically in man, meticulous edge-to-edge grafts seem advisable, as does the handling of postoperative leakage by suction drainage.

3. *Autografts of fascia, pleura, and pericardium with artificial support:* Because these tissues tend to fibrose and shrink rather than survive as inserted, and because of their tendency to split, separate, and leak when sutured, they are considered vastly inferior to dermal skin. The proper insertion of a supportive wire, as used in dermal grafts, is considerably more difficult; consequently, other forms of support have been more popular, such as wire gauze,[8] and mesh.[9] Such structures may deter healing because of their bulk; they probably afford more support than necessary, and in case of failure would be more troublesome than a length of thin wire coursing from side to side or disposed in a coil. Belsey[10] has reported two cases of intrathoracic tracheal excision for tumor, in which a longitudinal strip of trachea was not removed, and the defect ingeniously repaired with an envelope of fascia lata sutured over a large coil of stainless steel wire. Although these excisions were not complete sleeve resections, they could have been; and they were radical. This writer credits Belsey with the first successful, radical excision of the thoracic trachea, and feels a sincere admiration for his pioneering clinical work.

The original size of the reconstructed lumen in each of Belsey's cases was very large, evident from the diameter of the wire coils in reproduced roentgenograms, and from diagrammatic illustrations, so that, despite subsequent fibrous shrinkage of the fascial grafts, a functional tracheal lumen persisted in each instance. The construction of a very large lumen, when dealing with structures smaller than the trachea, is not always feasible, and fibrous contracture is likely to prevent such fortunate results.

The support of tubular grafts of these tissues with an indwelling tubular prosthesis has no apparent advantage over the use of the prosthesis alone, especially if the graft is converted to fibrous tissue, such as the fibrous tube the mediastinum will form about a foreign body.

The end result, in either case, is a fibrous cylinder connecting parts of the tracheobronchial tree. As long as a tube remains in its proper place, all may be well. If it becomes displaced, or has to be removed, the fibrous tissue, given sufficient time, is likely to assert its inherent qualities.

III. *Homografts With and Without Support.*—

If homogenous "split thickness" skin will not permanently "take" to ideal granulating surfaces, it seems illogical to expect other homogenous tissues to do so, and remain viable. The literature of the past, well presented by Jackson and associates and their own significant experiments,[11] is highly acceptable as conclusive evidence that the ultimate fate of homografts is usually fibrous

tissue and absorption. Therefore, whether or not a supporting prosthesis is used, at the present time, these grafts belong in a category similar to the one immediately preceding.

Limitation to the present is mentioned because of faith in the future efforts of our biologic and physical chemists. The delayed failure of homografts suggests loss of the function of cellular replacement or growth, possibly due to an unharmonious nuclear arrangement of individual cells. Perhaps gene patterns can be altered, or made harmonious, by the action of enzyme proteins (hormones, genes, vitamins). Clinically, at least, the "take" of an homogenous skin dressing can be markedly prolonged by ACTH. If the mode of action of such a protein is dependent, as acetylcholine is dependent,[12] on a "lock and key" fit of its nuclear pattern with its receptor substance, perhaps modern research will some day provide substances that suppress, or alter gene reactions, or other reactions, that are theoretically responsible for the incompatibility of homografts.

IV. *Prostheses Without Grafts.*—

This method has many attractions. Any type, size, or shape of tube can be supplied in unlimited quantity. Their use permits extraordinary experimental excisions. There is no particular need for the keen surgical judgment required when autobronchial grafts are contemplated. Technically, their use in animals is moderately simple. However, the clinical cases reported by Clagett and associates,[13] and mentioned by Jarvis,[14] suggest that this might not be true in man. The most attractive feature is the possibility of regeneration of tracheobronchial structures, including cartilages, as reported by Daniels[15] in his experiments.

On the other hand, the experimental failure of cartilage or tracheal regeneration about polythene tubing,[16] and in homografts,[11] and particulary its conspicuous rarity in man, in the fibrous, healed walls of stenosed bronchi, are noncorroborative evidences of such regeneration. In a study[6] of healed bronchial cicatrices, the failure of cartilage regrowth, subsequent to partial inflammatory destruction, within its natural environment, was regarded as one of the major factors in the pathogenesis of bronchostenosis. If, in man, regeneration fails in such situations, the hope for regrowth of tracheobronchial structures about a clinical prosthesis seems illogical.

It is felt that prostheses afford good temporary replacements, despite their lack of elasticity, and tendency to leak if he'd in place with encircling ligatures, and that their main drawback is the fibrous layer formed about them, which, as mentioned previously, is prone to cause trouble if the prosthesis is removed, or becomes displaced.

Actually, in the author's opinion, this very drawback offers a theoretical solution to a most difficult therapeutic problem. The fibrous structure produced by a foreign body tube, except for its lining, is little different from the fibrous tracheobronchial structures that have been successfully repaired with dermal grafts. The therapeutic problem in mind is that of extensive destructions and new growths of the trachea demanding complete, lengthy excisions.

In such instances, the necessary excision followed by the insertion of a suitable prosthesis is considered advisable. After the formation of a fibrous tube about the prosthesis, it could be removed, and subsequent contraction, or troublesome nonrigidity, minimized by the insertion of a well-rounded and heavily supported dermal graft.

CONCLUSIONS

1. Tracheobronchial reconstruction is clinically feasible and reasonable surgery. Its further development is necessary to continue the progress of pulmonary surgery.

2. The best reconstructive method is excision and anastomosis. Such anastomoses ordinarily do not need indwelling prostheses. Its use is facilitated if partial pulmonary resection is also necessary. It may be inadequate for extensive tracheobronchial lesions.

3. Of the available autografts, wire-supported dermal skin is considered the best, its clinical usefulness has been proved, and its supply is adequate. Bronchial autografts are ideal, but their use is limited by their availability, and unique clinical circumstances.

4. At the present time, homografts are unsatisfactory. The products of biochemical and biophysical research of the future may sometime make them successful.

5. Tubular prostheses are available, simple, and applicable in practically all tracheobronchial lesions. They provoke the formation of a fibrous cylinder about them, and their use may be only temporarily successful. Therefore, their replacement with proper dermal grafts is considered theoretically advisable, if they become displaced or have to be removed.

REFERENCES

1. Jackson, T. L., Lefkin, P., Tuttle, W., and Hampton, F.: An Experimental Study in Bronchial Anastomosis, J. THORACIC SURG. **18:** 630, 1949.
2. Gebauer, P. W.: Plastic Reconstruction of Tuberculous Bronchostenosis with Dermal Grafts, J. THORACIC SURG. **19:** 604, 1950.
3. Gebauer, P. W.: Further Experiences with Dermal Grafts for Healed Tubcerulous Stenosis of the Bronchi and Trachea, J. THORACIC SURG. **20:** 628, 1950.
4. Daniel, R. A., Jr., Taliaferro, R. M., and Schaffarzick, W. R.: Experimental Studies on the Repair of Wounds and Defects of the Trachea and Bronchi, Dis. Chest **17:** 426, 1950.
5. Carter, M. G., and Strieder, J. W.: Resection of the Trachea and Bronchi, an Experimental Study, J. THORACIC SURG. **20:** 613, 1950.
6. Gebauer, P. W.: Tuberculous Bronchostenosis: Pathology and Treatment, Hawaii M. J. **10:** 423, 1951.
7. Gebauer, P. W.: Experiences with Surgical Reconstruction of the Trachea, Am. Rev. Tuberc. **62:** 176, 1950.
8. Rob, C. G., and Bateman, G. H.: Reconstruction of the Trachea and Cervical Esophagus, Brit. J. Surg. **37:** 202, 1949.
9. Burnett, W. E.: Discussion of Papers on Tracheobronchial Grafts, J. THORACIC SURG. **20:** 650, 1950.
10. Belsey, R.: Resection and Reconstruction of the Intrathoracic Trachea, Brit. J. Surg. **38:** 200, 1950.
11. Jackson, T. L., O'Brien, E. J., Tuttle, W., and Meyer, J.: The Experimental Use of Homogenous Tracheal Transplants in the Restoration of Continuity of the Tracheobronchial Tree, J. THORACIC SURG. **20:** 598, 1950.
12. Welsh, J. H.: Structure Activity Relationship of Acetylcholine and Receptor Substance, Am. Scientist **38:** 239, 1950.
13. Clagett, O. T., Grindlay, J. H., and Moersch, H. J.: Resection of the Trachea, an Experimental Study and Report of a Case, Arch. Surg. **57:** 253, 1948.

14. Jarvis, F. J.: Discussion of Papers on Tracheobronchial Grafts, J. THORACIC SURG. **20**: 649, 1950.
15. Daniel, R. A., Jr.: The Regeneration of Defects of the Trachea and Bronchi. An Experimental Study, J. THORACIC SURG. **17**: 335, 1948.
16. Grindlay, J. H., and Mann, F. C.: Surgical Uses of Polythene; an Experimental Study, Arch. Surg. **56**: 794, 1948.

DISCUSSION

DR. DONALD L. PAULSON, Dallas, Texas.—Dr. Gebauer has demonstrated the superiority of wire-enforced dermal grafts for the plastic reconstruction of the trachea and bronchi. He has used it to reconstruct a portion of the circumference. In my own limited experience I have found wire-enforced dermal grafts to be entirely satisfactory. I have used this method in one instance in which the bronchus had ruptured and later underwent occlusion. I was able to reconstruct a satisfactory lumen. I have used it also in two tracheal cases, one a trachea involved by carcinoma, and the other a tuberculous stricture.

To reconstruct long lengths of the entire tracheal circumference, either fascia or dermis may be used in connection with steel wire or tantalum gauze. This method is probably preferable to the use of rigid tubes made of foreign substances such as polythene, vitallium, glass, lucite, and so forth. The steel wire or tantalum gauze provides the necessary rigidity and the dermis or fascia provides, as Dr. Gebauer has demonstrated in the case of dermis, a living scaffold for the ingrowth of epithelium. The tracheobronchial epithelium grows along the inner surface of the graft. In the case of hollow tubes of a foreign substance the epithelium grows outside the tube and the patient does not have the benefit of the tracheobronchial epithelium on the inside. The tracheal wall which does regenerate in the patients who have been reported is apparently not rigid enough to permit removal of the tube at a later date, and the patient is left with this tube or foreign substance unlined by epithelium. Robb and Bateman have demonstrated in the case of reconstruction of 9 cm. of trachea in which they used tantalum gauze wrapped with fascia, an ingrowth of epithelium on the inner surface. In using the same procedure to reconstruct an esophagus, they placed the fascia on the outside, on the outer surface of the tantalum gauze, and obtained a growth of epithelium on the outer surface, external to the tube. For that reason, I feel that the use of dermis with steel wire or tantalum gauze is probably preferable to the hollow tubes of foreign substance which have been used experimentally.

DR. LEWIS BOSHER, Jr., Richmond, Va.—I would like to report an experience with the Gebauer type of dermal graft applied to the trachea in conjunction with pneumonectomy. The difficulty of adequately supporting and vascularizing a dermal graft under these conditions, that is, when the lung has been removed, is obviously increased.

(Slide.) This is a 54-year-old white man who was admitted to our hospital after three months' study in a tuberculosis sanatorium where the diagnosis of tuberculosis was not confirmed. These are preoperative slides showing an abscess in the right upper lobe distal to a bronchial obstruction. At bronchoscopy a positive biopsy for carcinoma was obtained from the region of the right upper lobe bronchus. At thoracotomy it appeared that this carcinoma was limited to the region of the upper lobe bronchus and the main-stem bronchus, and that the lesion was resectable. The main-stem bronchus was divided one cartilage ring distal to the carina. The mucosa at that point appeared quite roughened, and a frozen section removed from the line of transection was interpreted by the pathologist as showing carcinoma in the mucosa. Accordingly a skin graft was prepared from the back, and threaded with No. 28 stainless steel wire. The bronchial stump was reopened and the bronchial orifice was amputated through the tracheal wall, removing the carina. It appeared that any attempt at direct closure of the defect would lead to marked tension and constriction. Accordingly, the dermal graft was sutured into place, turning the epidermal surface into the lumen.

(Slide.) The graft was sutured with No. 30 cotton. Partial coverage was obtained by displacing the azygos vien over the region and also by utilizing pericardial tissue. Complete coverage was not obtained. The pathologic report appeared to justify our attempts at a more radical approach to the problem; only one positive node was found in the resected specimen and this was within the lung tissue.

(Slide.) This shows the additional portion of bronchus resected. Frank carcinoma is seen in the left and to the right squamous metaplasia. A small additional safety margin was thus provided. Postoperatively the patient did satisfactorily except that he had considerable difficulty raising secretions and frequent bronchoscopies were required. At bronchoscopy there appeared to be rather considerable collapse of the graft into the lumen of the trachea on expiration, although on inspiration an entirely adequate lumen was present. On the fourteenth day, however, the patient began coughing up pleural fluid, and the pleural fluid had to be evacuated. Bronchoscopy showed a one millimeter fistula at the proximal edge of the graft. With the pleural space empty the graft no longer collapsed inward on expiration. Thoracoscopy showed that the graft was approximately tow-thirds granulated over. The graft was followed with frequent bronchoscopies and the latest was done several days ago, approximately five weeks after the operative procedure and three weeks after establishment of the fistula. At this time there was a 5 millimeter fistula at the proximal edge of the graft. The surface of the graft, which previously had appeared grayish white, was now replaced by a firm wall of pink granulation tissue which appeared well aligned with the wall of the trachea. We have hopes that this defect of the trachea will close over. The empyema which occurred after establishment of the fistula will have to be dealt with. It seems that more adequate coverage for such a graft will be essential if it is to be successful when employed in conjunction with pneumonectomy.

(Note: Six weeks following operation two-thirds of the dermal graft was coughed up by the patient, leaving a 1.5 cm. defect. The portion of the graft along the floor of the trachea remained intact and well epithelized.)

DR. MARK M. RAVITCH, Baltimore, Md.—What is essentially the method of Robb and Bateman reported in the *British Journal of Surgery* has been used in reconstruction of the trachea in two instances in our clinic, and in reconstruction of the esophagus in one instance. The first was a patient of Dr. Richard Kieffer—the reconstruction of a cylindrical segment of the trachea for carcinoma. The reconstruction was made over tantalum mesh covered on its outer side with a thin split thickness dermis graft, just the superficial layers of the dermis without the epidermis. About twelve days later the patient, apparently quite well, suddenly expired from hemorrhage due to erosion of the carotid artery by a loose fragment of the tantalum mesh.

The second case, a patient of Dr. Jacob Mandlesman, had a carcinoma of the trachea involving a portion of the right main bronchus. All but a 5 millimeter strip of the trachea posteriorly was resected, and a portion of the right main bronchus, and the same type of reconstruction was performed, entirely successfully. That patient left the hospital with complete epithelization. It might be added that in both cases there was a cervical tracheotomy to aid the anesthesia, and anesthesia on the operative side was maintained during operation by another tube passed in to the operated lung going over the side of the table to another machine.

The third instance of tantalum mesh repair was by Dr. Milton Edgerton, a reconstruction of the esophague and, in this case, as Dr. Paulson intimated, the tantalum mesh was later removed, epithelization having occurred on the outside of the mesh.

DR. WILLIAM CAHAN, New York.—(Slide.) This is an 18-year-old man who had a history of recurrent bouts of pneumonitis for a period of six years, and finally had his first hemoptysis. Bronchoscopy revealed that the lumen was narrowed to about 40 per cent of its normal size. A biopsy of the tissue showed an adenoid cystic carcinoma, salivary gland type. The tracheogram shows the filling defect and the lateral view shows the defect having its inferior margins 32 millimeters above the carina.

(Slide.) The patient was placed in the right lateral position and the right chest was opened and the tumor identified. The trachea was then completely mobilized; the vagus nerve retracted.

(Slide.) This is a facsimile of the tantalum gauze-fascia lata graft, according to the technique of Rob and Bateman. The graft was tacked at many points along the mesh itself to prevent slipping.

(Slide.) The tumor has been removed and the intratracheal tube passed distal to the defect and into the opposite bronchus.

(Slide.) This is the defect after excision of the tumor. Tantalum mesh with the fascia lata backing was then sewn so that tantalum gauze faced the lumen.

(Slide.) This shows the final closure with a pleural flap raised at the onset of the procedure, used to cover the entire field.

(Slide.) A tracheotomy was done at the conclusion of the procedure for two purposes: one, it served as an excellent point of suction; and two, we reasoned that perhaps it might be a decompression point for postoperative coughing, relieving the suture line of the force of down draft from vocal cord occlusion.

(Slide.) The lateral view shows that there has been no diminution of the size of the lumen.

(Slide.) This is the tumor, measuring 3 by 2 by 2 centimeters. The tracheal defect measured about 7 by 3 centimeters. It is now six months after operation and we bronchoscoped him last week. Other than some tufts of granulation tissue, there was no evidence of disease.

DR. GEBAUER (closing).—I would like to thank the discussers. In regard to tracheal reconstruction, there is a saying that the proof of the pudding is in the eating. We have recently been served a most delicious banquet along these lines. We have a patient, whom my medical colleagues regard as a surgical miracle, and whom I regard as a most difficult job with a good result, almost unbelievable to date.

(Slide.) In this patient, I watched a tracheobronchial tuberculosis extend and heal for a period of two years. The ultimate result was complete occlusion of the intermediate bronchus on the right, with atelectasis of the middle and lower lobes; you can see a small, dense, wedge in the costophrenic angle on the right, with severe stenosis of the entire left main bronchus and the lower two-thirds of the trachea.

(Slide.) This shows the progression in stenosis of the trachea over a period of six months, from February to July, 1950. Despite dilatations, the stenosis progressed, and in November, 1950, a tracheotomy had to be done. In order to get a tracheotomy tube through and beyond the stenosis of the trachea it was necessary to forcibly rupture the trachea and put in a long cane-shaped tube.

(Slide.) This is after tracheotomy; here is the end of the tracheotomy tube, extending from the neck to the bifurcation. The patient has had thoracotomy on this side and the entire left bronchus was reconstructed with a dermal graft. Seven weeks later, we operated on the right side and removed the right middle and lower lobes. We were able to pull down the intratracheal tube and place it in the grafted left bronchus. It was difficult, grossly, to tell which part of the bronchus had been grafted. We then repaired the trachea, with a second dermal graft, so that this patient has now a bronchial graft extending from the level of the upper lobe bronchus on the left to beyond the carina, and from the carina into the cervical region.

(Slide.) These are the entire middle and lower lobes. This is the area in the middle lobe that was probably the source of the positive sputum.

(Slide.) This was taken two months after operation on the right side, and about six weeks after removal of a short, standard tracheotomy tube. This patient has been relieved of her chronic asphyxia and is sputum negative. If her present good clinical state persists, she will be discharged in the near future.

I believe that it should be mentioned that the bronchoscopic observations relative to reconstructive tracheobronchial procedures are a part of the surgeon's job. In fact, I shhould feel severely handicapped, if not completely lost, if I were not able personally to visualize the active, the healing, and the constrictive phases of these lesions, and evaluate them bronchoscopically before and after operation.

MYXOCHONDROMA OF THE TRACHEA

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A CASE REPORT

PRIMARY tumors of the trachea are rather uncommon, and the literature contains but few reports of direct surgical excision. A variety of methods have been used to restore or preserve the continuity of the trachea, such as metal and plastic tubes and fascia supported by tantalum gauze on loops of wire. Gebauer,[1, 2] in a discussion of the use of dermal grafts for the reconstruction of tuberculous stenotic bronchial and tracheal lesions, predicted that this method might well find use in the management of acute, traumatic, tracheobronchial defects and those resulting from surgical excision.

Recently I had the opportunity to manage a patient with a benign obstructing lesion of the trachea by the use of a wire-enforced full thickness skin graft. Recovery was uneventful, with restoration of the normal contour of the trachea and the preservation of voice. The case is presented:

N. W. was a white man aged 59 years. He had noted the onset of a wheeze during the winter of 1949 which had slowly become progressive. In September, 1950, the wheeze had become marked and was associated with cough and mucopurulent sputum. Transient periods of dyspnea had appeared which were relieved in part by spasms of coughing and the raising of mucous plugs. Blood-streaked sputum had been present on two occasions. There was no chest pain or loss of weight associated with the illness. By Oct. 1, 1950, the patient was forced to discontinue his activities as a druggist because of shortness of breath. He was referred* to me because of a tracheal tumor seen on indirect laryngoscopy.

Upon physical examination on Oct. 19, 1950, the patient was a slender, rather well-developed man who appeared neither acutely nor chronically ill. The positive findings were limited to a marked inspiratory and expiratory wheeze which was heard over the anterior neck immediately below the larynx. The trachea was freely movable in the neck and there was no lymphadenopathy present. Roentgenographic examination of the chest was not remarkable. Indirect laryngoscopy revealed the vocal cords to move normally, and a large tumor mass was visualized within the trachea, appearing to arise from the left lateral wall of the trachea and its posterior portion. The trachea airway was reduced to a concentric opening anteriorly and to the right. Tomograms of the neck had revealed a circumscribed tumor mass within the trachea which appeared to arise at the level of the first tracheal ring with encroachment upon the cricoid cartilage.

The patient was admitted to the hospital on Oct. 21, 1950, and at that time, under local anesthesia with pontocaine, the 5 mm. × 30 cm. full lumen Jackson bronchoscope was introduced directly past the tumor into the lower trachea. A low tracheotomy was done through a transverse incision. It was felt that with the tracheotomy tube in place, biopsy of the tumor mass could be more safely carried out, particularly in reference to the management of any bleeding that should occur. The biopsy† revealed a myxochon-

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*By Dr. Jewell Smith and Dr. Samuel Ross, Fresno, Calif.

†By Dr. J. Bocian, Fresno County Hospital, Fresno, Calif.

droma of the trachea without evidence of malignant changes. The patient was discharged from the hospital on Oct. 24, 1950.

It was felt, from examination of the roentgenograms (Fig. 1), the tumor might well be locally excised by direct surgical approach. As the consequence, the patient was readmitted and on Nov. 2, 1950, under intratracheal anesthesia through the tracheotomy opening, and the neck was explored. The left lateral wall of the trachea was approached by a dissection between the lateral lobe of the thyroid and the carotid sheath. The fixation of the lower trachea by the tracheotomy wound prevented rotation of the trachea

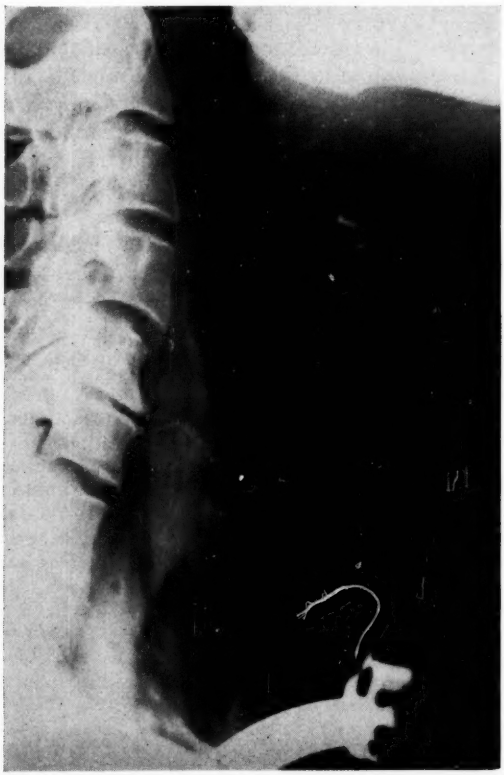

Fig. 1.—Left lateral view of the neck showing the large tracheal tumor from the first tracheal ring and the cricoid cartilage. The tracheotomy tube is in place.

and esophagus forward. As a consequence, the lateral wall of the trachea was exposed. The recurrent nerve was carefully preserved. The second and third tracheal cartilages were divided posteriorly at the junction with the membranous wall, and the tumor mass encountered.

The tumor mass measured approximately twelve millimeters at its greatest diameter and almost completely filled the trachea in the subcricoid area. It appeared to arise from a base approximately one centimeter in diameter from the first tracheal cartilage and also from the cricoid cartilage. Approximately one-half of the first tracheal cartilage, one-third of the second tracheal cartilage, and one-fourth of the third tracheal cartilage, together

with a portion of the left lateral and posterior wall of the cricoid cartilage, were removed with the tumor. The surgically created defect measured approximately 3 by 1½ centimeters.

In preparation for the surgical removal of the tumor, several stainless steel tubes with a flange at either end had been constructed. However, none of the tubes appeared to be suitable for reconstruction of the trachea. A full thickness skin graft was taken from the inner aspect of the right arm and reinforced with No. 28 gauge stainless steel wire after the manner of Gebauer.[1, 2] The subcutaneous tissue was carefully dissected from the deep surface of the graft. The wire supported graft was then bent in a slight curve and placed in the defect of the trachea. It was sutured in place with interrupted fine stainless steel wire so that an airtight closure of the trachea was accomplished.

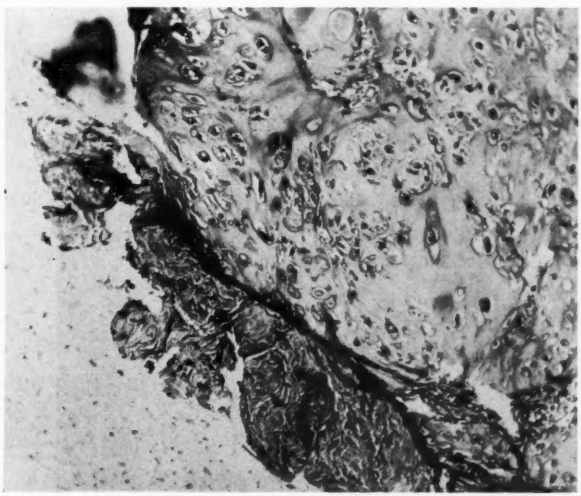

Fig. 2.—Photomicrograph ×100 of the tumor showing the immature cartilage cells.

A fine Penrose drain was inserted into the lower angle of the wound adjacent to the trachea. The wound was closed in anatomic layers using fine interrupted silk sutures. The subcutaneous tissue was carefully approximated with interrupted sutures of fine silk. The skin was left open. The donor site of the right arm was closed with interrupted black silk sutures.

The patient was placed on penicillin therapy postoperatively, and recovery was entirely uneventful. The Penrose drain was removed on the second postoperative day. There having been no evidence of subcutaneous emphysema throughout the postoperative stage, the patient was discharged to his home on the fourth postoperative day with the tracheotomy tube in place.

He was admitted again on Nov. 20, 1950. At this time the wound was well healed. The examination of the larynx by direct laryngoscopy revealed the left arytenoid process to be somewhat distorted and the left vocal cord to be adducted and fixed. The trachea airway appeared widely patent, and the right vocal cord moved normally. The 7 mm. × 40 cm. bronchoscope was introduced into the trachea, and the graft was readily apparent. It appeared pinkish-white in color, and there was a tiny area of granulation tissue along the medial margin of the graft adjacent to the junction with the membranous wall of

the trachea. The remaining portion of the trachea appeared entirely normal. The patient was again discharged the following day, and on Nov. 23, 1950, the tracheotomy tube was removed. The tracheal wound promptly closed.

Pathologic examination of the tissue removed revealed a well-encapsulated tumor measuring approximately 2 by 1½ centimeters. The outer surface was smooth and seemingly well encapsulated. The underlying tissue was gray-tan and of moderate consistency to palpation. Several tiny sections of cartilage were attached to the tumor mass. Microscopic examination of the tumor proper (Fig. 2) revealed a normal tracheal epithelium with a chronic inflammatory infiltration in the submucosa. There were sections of poorly developed cartilage intermixed with a bluish matrix in which were found isolated star-shaped cells. The pathologic diagnosis* was a myxochondroma of trachea.

Fig. 3.—Postoperative film showing the wire enforced graft in place and the normal anterior posterior diameter of the upper trachea.

Recovery has been excellent. The patient was seen in follow-up on April 9, 1951 (Figs. 3 and 4), at which time laryngoscopy revealed both vocal cords to move normally, and the trachea was widely patent. Tracheoscopy revealed that the graft was well healed and normal in appearance. There was no evidence of granulation tissue or scarring of the tracheal wall. The tracheal contour was normal. Roentgenographic examination of the neck revealed normal anterior and lateral diameters of the trachea. The wire within the

*By Dr. J. Bocian, Fresno, Calif.

graft remained unchanged in its position. The patient has continued to be asymptomatic, free of cough and sputum, and appears to be enjoying excellent health. He has returned to his former occupation as a pharmacist.

DISCUSSION

It is felt that this patient is of interest insofar as primary neoplasms of the trachea are infrequently encountered. In the past, these tumors have been treated by bronchoscopic removal. However, in a few instances, neoplasms of the trachea have been directly attacked by surgical incision; as in the case of Jarvis[3] in which he removed a cylindroma of the intrathoracic portion of the trachea and restored continuity by a stainless steel tube.

Fig. 4.—Postoperative view of the neck with the graft in place to the left of the midline in the upper trachea.

Clagett and associates[4] excised a cylindroma of the trachea, and used a prosthesis of plastic material for repair.

Rob and Bateman,[5] in the treatment of a recurrent thyroid carcinoma with involvement of the cricoid cartilage and upper trachea, resected the involved portion and restored continuity by the use of a tantalum wire mesh

gauze over which fascia lata was placed. Emphysema in the neck was a postoperative complication.

More recently, Belsey[6] reported two cases of cylindroma of the intrathoracic portion of the trachea in which surgical excision was carried out and restoration of continuity was performed by a fascia graft over wire loops.

In the patient reported, N. W., it is felt that a method by which the tumor might be excised by direct surgical approach would be much more desirable than by removal bronchoscopically. The method described by Gebauer,[1, 2] which has been used successfully by him in the management by tuberculous strictures of the trachea and the bronchi, appeared to be the most suitable for the management of this individual patient. The support given the trachea by the incorporation of a skin graft enforced by stainless steel wire into its wall was adequate to give airtight closure and rapid healing. The patient has remained well since the operation.

SUMMARY

A case is reported of a large benign tracheal neoplasm, a myxochondroma surgically removed. The repair of the tracheal defect was undertaken successfully by the incorporation of a full thickness skin graft enforced by a stainless steel wire.

ADDENDUM

The patient was last seen on Nov. 10, 1951, at which time the graft remained unchanged on roentgenogram, the trachea widely patent, and the patient asymptomatic without evidence of recurrence.

REFERENCES

1. Gebauer, Paul W.: Plastic Reconstruction of Tuberculous Bronchostenosis with Dermal Grafts, J. THORACIC SURG. **19**: 604-628, 1950.
2. Gebauer, Paul W.: Further Experiences with Dermal Grafts for Healed Tuberculous Stenosis of the Bronchi and Trachea, J. THORACIC SURG. **20**: 628-647, 1950.
3. Jarvis, F. J.: Discussion of Reference 2. J. THORACIC SURG. **20**: 649, 1950.
4. Clagett, O. T., Grindlay, J. H., and Moersch, Herman J.: Resection of the Trachea, An Experimental Study and Report of a Case, Arch. Surg. **57**: 253, 1948.
5. Rob, C. G., and Bateman, G. H.: Reconstruction of the Trachea and Cervical Esophagus, Brit. J. Surg. **37**: 202, 1949.
6. Belsey, Ronald: Resection and Reconstruction of the Intrathoracic Trachea, Brit. J. Surg. **38**: 200-205, 1950.

DISCUSSION

DR. ALFRED GOLDMAN, Beverly Hills, Calif.—I want to apologize for discussing this paper because I have no tracheal tumor to report, but Dr. Evans did mention the direct approach to excision of tracheal tumors, and what I want to report is the direct approach to such bronchial tumors; that is, the local excision of bronchial tumors. This has been attempted in five instances, and in three it has been eminently successful. The amount of cartilage removed with each tumor varied: none in one, two rings in one, and five rings in another. In two cases the operation could not be done.

(Slide.) I wish to report the case of a 14-year-old girl. This shows the preoperative roentgenogram with complete atelectasis of the left lung. The symptoms had started some six months before, with wheezing, and the atelectasis was of short duration.

(Slide.) This is the planigram and shows the obstructing tumor close to the carina in the left stem bronchus.

(Slide.) This is a colored slide of the specimen that was removed.

(Slide.) This is the histologic appearance of this tumor, a glandular bronchial adenoma serous in type.

(Slide.) This is a cross section of the tumor. This is a portion of the stalk. It was possible to open the left stem bronchus to expose the entire area from the carina down. The tumor was about 4.5 centimeters in length and about 2 centimeters in diameter, and was attached by a stalk 0.5 centimeter in diameter over an area of about two rings, but it did not infiltrate beyond the rings. It was possible to excise the stalk and under direct vision to excise the mucosa around it and to treat the base with a high frequency cautery. It was possible to feel all around the bronchus, internal, external, and the subcarinal area, and there was no extension.

(Slide.) We studied the postoperative expansion of this lung, because if this operation is to be done we must be sure that the remaining lung will become at least ventilated and aerated, and not be the source of further trouble. Here you see that on the day of operation the lung is still collapsed and there is air in the pleura. Two days later there is some expansion, some fluid, and the lung is still opaque.

(Slide.) Ten days later the lung shows some aeration.

(Slide.) These are lipiodol studies done five weeks after operation. There is a cylindrical bronchiectasis involving several segments of the lower lobe, and club-shaped bronchiectasis which involves the posterior superior segment, seen in the lateral view.

(Slide.) This is a lipiodol study made thirteen months after operation, and in the lateral view there is no longer any club-shaped bronchiectasis visible and very little cylindrical bronchiectasis in the other segments. This girl has remained well and free of symptoms for more than twenty-six months since operation.

ANALYSIS OF TWENTY-SEVEN CASES OF ACUTE CARDIAC ARREST

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IN RECENT years there has been considerable discussion of the condition called acute cardiac arrest by which is meant the sudden cessation of effective cardiac beat. It has as its immediate effect complete interference with the oxygen and carbon dioxide transport system. This terrifying condition is not rare even during minor surgical procedures, and often results in death of the patient on the operating table. There really exists no greater surgical emergency, and still this condition is poorly understood by most surgeons and certainly inadequately treated in many instances. Unless the understanding of this urgent surgical emergency is clear a regimen for rapid, effective treatment is not possible.

During the last ten years there have been at the University Hospitals, State University of Iowa, twenty-five instances of acute cardiac arrest in approximately 71,000 anesthetic and operative procedures. Two additional cases are included which occurred in poliomyelitis patients who did not receive anesthetics during the acute cardiac arrest. This makes an incidence of 0.35 per 1,000 of anesthetics given. Nicholson[1] reports seven cases of cardiac arrest in 3,500 anesthetics, and Trent[2] reports twenty-seven instances in 40,000 anesthesias and operative procedures. This accident can occur without regard to the type of operative procedure performed, anesthetic agent used, or the age or general physical state of the patient.

Until about four years ago what is now considered the most efficacious, immediate treatment had not been instituted in our cases. The physician anesthetist who maintains close contact with the changing physiologic state of the patient is the first of the surgical team to note the presence of cardiac standstill. The course of treatment must be initiated without delay in order to combat the immediate asphyxia which occurs in the brain and the heart. It is known that the maximum time during which circulation may be allowed to be inadequate is between three and three and a half minutes under the most optimum conditions and in the absence of previous chronic anoxia.[3, 4] The brain will not tolerate asphyxia for a longer period of time and recuperate normally from this acute insult. The primary consideration after cardiac arrest has occurred is the prompt provision of artificial circulation to transport oxygen to the tissues and to eliminate carbon dioxide from the tissues.

The first group of eleven cases of cardiac syncope which occurred between 1942 and 1947 are tabulated in Table I. In this group there were no survivals.

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The method of therapy during this time consisted only of intravenous or intracardiac injection of medications and artificial ventilation. The second group of sixteen cases which occurred between 1948 and 1951 are presented in Table II. In this group of patients attempts at artificial circulation were made, resulting in varied lengths of survival. Three patients are alive at the present time, one of whom shows considerable cerebral damage. One additional patient survived fifteen days and was apparently well at the time of discharge from the hospital, but died suddenly after returning home.

The major difference between the treatment in the two groups of patients is the addition of the use of artificial circulation. Criticism must be made that there has often been a much too great lapse of time between recognition of the emergency state and the institution of effective artificial circulation. Because of this many of these patients did die. Some survived many hours following the acute emergency, and the deaths were due to irreversible cerebral damage.

TABLE I*

NO.	AGE	PHYSICAL STATE	PROCEDURE	TREATMENT	SURVIVAL	ANESTHETIC AGENT
1	8 wk.	Good	Harelip repair	Intracardiac Epinephrine	0	Ether
2	10 yr.	Fair	Pneumonectomy	Intracardiac Epinephrine Coramine	0	Cyclopropane and Ether
3	51 yr.	Poor	Colostomy	—	0	Cyclopropane
4	39 yr.	Excellent	Cholecystectomy	Tracheotomy	0	Cyclopropane
5	6 wk.	Poor	Cerebellar exploration	—	0	Ether
6	65 yr.	Poor	Smith-Petersen nailing	Intracardiac	0	Nitrous oxide and pentothal
7	48 yr.	Excellent	Dilatation and curettage	—	0	Cyclopropane
8	6 yr.	Fair	Skin graft	Intracardiac Adrenaline, Procaine Coramine	0	Cyclopropane
9	7 wk.	Good	Harelip repair	—	0	Chloroform
10	22 mo.	Good	Cleft palate repair	—	0	Chloroform
11	26 yr.	Poor	I & D leg abscess	—	0	Nitrous oxide and pentothal

*All had artificial ventilation.

CASE REPORTS

CASE 1.—This 51-year-old white woman was admitted with the complaint of difficulty in swallowing of seven months' duration. She was known to have had diabetes for seventeen years and known to have severe hypertensive cardiovascular disease. Two cerebral vascular accidents had occurred within the last four years, resulting in some permanent weakness of the left arm and leg. Physical examination revealed a blood pressure of 200/90 and moderate weakness of left arm and leg. Esophagoscopy was unsuccessfully attempted under local anesthesia. A second attempt of esophagoscopy was done four days later under general anesthesia using ether. At the time the esophagoscope was inserted the pulses suddenly became nonperceptible, and respirations stopped. Artificial ventilation was started immediately, and artificial circulation through a transabdominal approach was made within six minutes

TABLE II*

NO.	AGE	PHYSICAL STATE	PROCEDURE	ARTIFICIAL CIRCULATION		APPROACH TO HEART	SURVIVAL	ANESTHETIC AGENT	REMARKS
				INTERVAL TO ONSET	DURATION				
1	51	Poor	Esophagoscopy	6 min.	1 min.	Transabdominal	80 hours	Ether	Overdosage
2	8	Fair	Left thoracotomy	½ min.	34 min.	Transthoracic	Alive and well	Nitrous oxide and nembutal	Anoxia due to aspiration
3	79	Good	Cholecystojejunostomy	½ min.	1 min.	Transdiaphragmatic	Discharged 15th PO day†	Nitrous oxide and pentothal	—
4	6	Poor	I. V. Angiocardiography	1 min.	10 min.	Transthoracic	5 hours	Nitrous oxide and pentothal	Tetralogy of Fallot
5	63	Poor	Gastrojejunostomy	16 min.	4 min.	Transdiaphragmatic	70 hours	Nitrous oxide and nembutal	Cerebral damage
6	23	Good	Ligation patent ductus arteriosus	1 min.	120 min.	Transthoracic	2 hours	Nitrous oxide and demerol	Massive hemorrhage, fibrillation, electric shock
7	70	Fair	Ureterointestinal anastomosis	4 min.	30 min.	Transdiaphragmatic	½ hour	Cyclopropane and nitrous oxide	Fibrillation
8	4	Good	Harelip repair	5 min.	20 min.	Transabdominal	0	Ether	Anoxia aspiration of blood
9	7	Excellent	Tonsillectomy	5 min.	2 min.	Transthoracic	Alive‡	Ether	Overdosage
10	9 mo.	Fair	Cerebellar exploration	3 min.	10 min.	Transthoracic	50 minutes	Nitrous oxide and nembutal	—
11	63	Good	Gastric resection	6 min.	45 min.	Transthoracic	12 hours	Nitrous oxide and demerol	Anoxia faulty intratracheal tube
12	68	Poor	Exploration of renal area	3 min.	150 min.	Transthoracic	2½ hours	Cyclopropane and nitrous oxide	Cardiac failure A.S. heart disease
13	7	Poor	Tracheotomy	3 min.	5 min.	Transthoracic	24 hours	None	Bulbar poliomyelitis
14	25	Poor	—	30 min.	40 min.	Transthoracic	12 hours	None	Poliomyelitis Aspiration of food
15	34	Fair	Left thoracotomy	½ min.	1 min.	Transthoracic	Alive and well	Nitrous oxide and pentothal	Anoxia due to aspiration
16	2	Good	Skin plasty	22 min.	5 min.	Transthoracic	0	Ether	Overdosage

*All had artificial ventilation.
†Died suddenly 2 days after discharge, cause unknown.
‡Residual cerebral damage.

following recognition of the acute cardiac arrest. One milligram of epinephrine was injected into the heart. Artificial circulation was maintained for one minute, following which the cardiac beat was restored and blood presure could be obtained at a systolic level of 250 mm. Hg. Following this incident, the patient failed to regain consciousness. The patient developed a severe hyperthermia and expired eighty hours following the onset of the acute cardiac arrest. Autopsy findings revealed an inflammatory stricture in the upper esophagus, a healed area of infarction in the right cerebral hemisphere, a pneumothorax on the left with complete collapse of the left lung, and cardiac hypertrophy with old healed myocardial infarcts. It was felt that the immediate cause of death was due to the cerebral anoxia following the cardiac standstill.

CASE 2.—This 9-year-old boy was admitted with the complaint of a productive cough, intermittent fever of five years' duration. At the age of 3½ years the patient suffered an injury to his left hand which necessitated a general ether anesthesia for repair and amputation of part of the digits. This was followed by development of multiple lung abscesses and bronchiectasis in his left lung. Bronchograms at the time of admission revealed a completely destroyed left lung due to extensive bronchiectasis. A left pneumonectomy was attempted under nitrous oxide and nembutal anesthesia. It was noted that the patient was spilling large amounts of purulent material which had to be removed by suction at frequent intervals. Some short periods of cyanosis were noticed by the anesthetist at those times. During the procedure of freeing some of the adherent left lung from the chest cage, sudden cardiac arrest developed. The heart was under direct vision at that time and artificial circulation by manual compression of the heart was started within half a minute of recognition of the state of cardiac arrest. Artificial ventilation was given throughout this period. Artificial circulation had to be maintained for thirty-four minutes before the heartbeat became regular and blood pressures of 78/40 could be obtained. This patient failed to resume spontaneous respiration postoperatively and so was placed in a Drinker type of respirator for about seven hours. Forty-four hours following the operative procedure the patient became quite rational and apparently free of all abnormal neurologic findings. Bilateral Babinski sign had been present in the immediate postoperative period. Three days later the patient became ambulatory but appeared somewhat lethargic. Five days following the cardiac arrest the patient again became drowsy, and it was felt that there was definite visual impairment. This was followed by the development of generalized athetoid movements of both arms and legs. Seventeen days following the acute cardiac arrest all abnormal neurologic findings had disappeared, and the patient was discharged in what was thought to be normal condition. This patient re-entered the hospital one year after the previous acute episode, and a left pneumonectomy was carried out without any further difficulty. At that time the patient showed no abnormal neurologic findings.

CASE 3.—This 79-year-old woman was admitted with the complaint of jaundice of two weeks' duration. She gave a history of 30-pound weight loss and clay colored stools. A preoperative diagnosis of cholecystitis and cholelithiasis had been made. An exploratory laparotomy revealed an inoperable carcinoma of the head of the pancreas and a cholecystojejunostomy was performed. Anesthesia was provided by nitrous oxide, demerol, and nembutal. During the procedure the patient suddenly developed cardiac arrest. Artificial circulation was started within thirty seconds after cessation of the cardiac beat by passing the hand through a rent in the diaphragm. One hundred milligrams of procaine were given on two occasions intracardially. Good peripheral pulses were noted one minute after the artificial circulation had been started, and blood pressures of 180/80 and a pulse rate of 92 per minute were recorded. The operative procedure was then finished without incident. Electrocardiograms taken on the day of operation showed a normal sinus rhythm. Forty-eight hours following the operative procedure the patient developed an episode of acute left ventricular failure with some pulmonary edema which responded well to medication. On the third postoperative day the patient developed some cardiac arrhythmia which had disappeared by the tenth postoperative day. The patient was discharged on the fifteenth postoperative day in good general condition, but she expired suddenly forty-eight hours after return to her home.

CASE 4.—This 7-year-old boy was admitted for intravenous angiocardiographic studies. It was thought that this patient had a Tetralogy of Fallot and severe pulmonic stenosis. The patient had had episodes of cardiac failure twice in the preceding six years and had had digitalis two months before this admission. On physical examination the patient was a poorly nourished, extremely cyanotic 7-year-old boy. There was a harsh systolic murmur heard over the third interspace to the left of the sternum and a palpable thrill. The rhythm was regular. The blood pressure was 108/60. Cardiac fluoroscopy established a diagnosis of cyanotic congenital heart disease, probably Tetralogy of Fallot with a right descending aortic arch. Electrocardiograms showed a right bundle branch block and a very marked right ventricular hypertrophy. During the intravenous administration of 35 c.c. of neoiopax during nitrous oxide and pentothal anesthesia, the patient developed acute cardiac arrest. Artificial circulation through a transthoracic approach was started within one minute after onset and recognition of the acute cardiac standstill. Artificial circulation was maintained for ten minutes before an effective beat of 50 to 60 beats per minute was established. Artificial ventilation was maintained throughout. It was noticed that the patient developed a convulsive seizure involving the eyelids and the upper extremities bilaterally shortly after artificial circulation was begun. The patient developed hyperpyrexia and never regained consciousness following this acute episode and he expired five hours following the acute cardiac arrest. The postmortem examination was limited to the chest and established the diagnosis of a severe pulmonic stenosis and Tetralogy of Fallot with a right-sided aortic arch. Microscopic sections of the heart muscle showed some extravasation of blood into the cardiac musculature. The heart weighed 250 grams. It was felt that this patient died of the cerebral complications of anoxia.

CASE 5.—This 63-year-old white woman was admitted with the symptoms of intestinal obstruction. It was thought to be due to a neoplasm in the hepatic flexure of the colon, and an exploratory laparotomy was planned. The patient developed acute cardiac arrest seventeen minutes following the induction of the anesthesia with nembutal, curare and pentothal and following oral intubation and administration of cocaine to the pharynx. Artificial circulation was maintained for four minutes before adequate peripheral systolic blood pressures could be obtained. Shortly following the onset of the acute cardiac arrest the operative procedure was continued and a gastrojejunostomy was established for a duodenal obstruction which was found to be present. Twelve hours following the operative procedure the patient experienced a few convulsive seizures, generalized in character. She never regained consciousness following the surgical procedures. and expired seventy hours following the incident. Electrocardiographic tracings immediately postoperatively showed sinus tachycardia with some evidence of myocardial damage. Among the findings revealed by postmortem examination were diffuse degenerative changes of the brain probably due to the acute anoxia, thrombosis of the left middle cerebral artery, and multiple infarcts of the cerebral cortex.

CASE 6.—This 23-year-old man was admitted with the diagnosis of a patent ductus arteriosus. A left exploratory thoracotomy was carried out in an attempt to ligate the patent ductus. At the time of dissection it was found that the ductus was extremely friable and it tore spontaneously, necessitating rapid application of clamps across the structure to prevent severe hemorrhage. The anesthesia used at the time was nitrous oxide, demerol, and nembutal, with an endotracheal catheter in place. During the course of the procedure, uncontrollable hemorrhage from the pulmonary as well as from the aortic end of the ductus occurred, necessitating rapid transfusions to maintain the patient's blood pressure. The patient developed cardiac arrest probably due to a period of hypoxia accompanying this acute blood loss. One cubic centimeter of 1:10,000 epinephrine was injected which was followed immediately by ventricular fibrillation (Fig. 1). Artificial circulation was instituted immediately and had to be carried out for about two hours. Fibrillation continued. Within seven minutes of the onset of the ventricular fibrillation, a 110 volt alternating current was applied to the cardiac muscle which was followed by asystole for about two minutes then followed again by ventricular fibrillation. The ventricular fibrillation was converted twice more by application of the 110 volt alternating current, but the patient succumbed on the

operating table two hours after the onset of the cardiac arrest. Post-mortem findings on this patient revealed an extremely sclerotic and edematous patent ductus arteriosus with evidence of bacterial endarteritis and vegetations on the pulmonary site of the patent ductus.

CASE 7.—A 70-year-old man was admitted with a complaint of pain over the left side of the abdomen, flank, and in the suprapubic area. The patient had previously had an abdominal perineal resection performed for an adenocarcinoma of the rectum. Investigation at this admission revealed a mild uremia and a hydronephrosis on the left. It was felt that the patient had a recurrence of the neoplasm in the region of the left ureterovesical junction, but an exploratory laparotomy revealed only a large amount of scarring in the region of the bladder and at the left ureterovesical junction, and no evidence of recurrence of the rectal neoplasm. Three weeks later the patient was reoperated to perform a ureterovesical anastomosis. Anesthesia used was cyclopropane and nitrous oxide plus pentothal and demerol

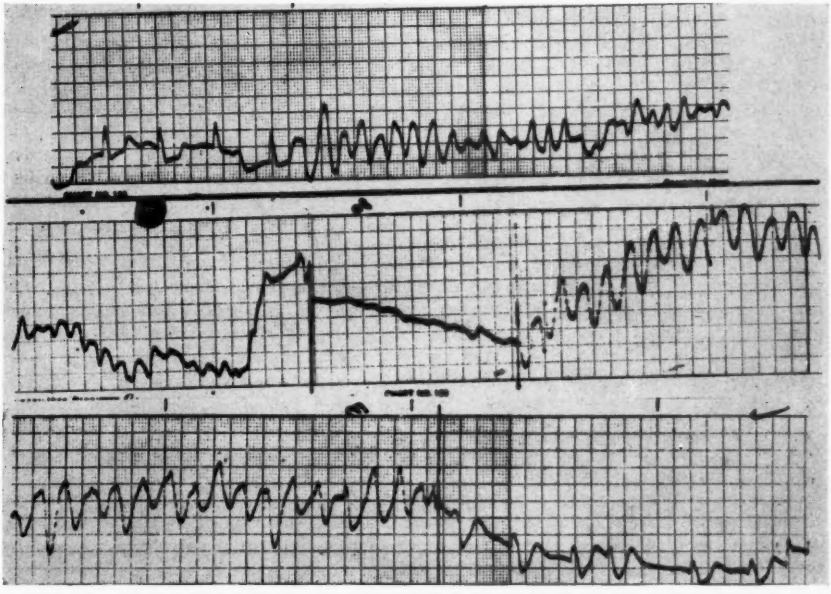

Fig. 1.—Shows occurrence of ventricular fibrillation asystole after electric shock; recurrence of ventricular fibrillation; shock again followed by bizarre ventricular rhythm.

and some curare. Sixty minutes after the start of the anesthesia sudden cardiac arrest occurred. Artificial circulation was instituted four minutes following the onset of the incident through a transabdominal, transdiaphragmatic approach. 0.5 mg. of epinephrine was injected into the right auricle which was followed by ventricular fibrillation. Injection of procaine did not change the fibrillation. The heart failed to respond. Within a few minutes fibrillation ceased and the patient was pronounced dead, thirty-four minutes following the sudden loss of pulse and blood pressure.

CASE 8.—This 4-year-old girl was admitted for a posterior cleft palate repair. The child had had previous operations for repair of a harelip and cleft palate. During this procedure the anesthesia, with open drop vinethene for induction, was followed by open drop ether. Two minutes after the start of administration of ether the patient ceased breathing. Artificial respiration was started immediately with a breathing bag mask and 100 per cent oxygen. The patient recovered with no apparent untoward effects. The anesthetic was

again started in a similar manner using open drop vinethene and then changing to ether. The operation proceeded for about twenty minutes with frequent interruptions because of bleeding which caused respiratory obstruction and necessitated suction. Thirty minutes after the start of the operation the patient seemed again to become obstructed. At this time the anesthetist was unable to obtain any peripheral pulse or elicit evidence of cardiac action. Artificial respiration was instituted immediately. There was no obstruction and the lungs inflated easily, but the patient continued to be cyanotic. A bronchoscope was introduced, but no significant secretions could be seen. One milligram of intracardiac adrenalin was given at the surgeon's suggestion. For about thirty seconds the patient's pulse was audible, and cyanosis improved. About five minutes after the cessation of respiration artificial circulation was instituted through a transabdominal incision. This was continued for about twenty minutes with no apparent recovery of cardiac activity. Fifty-five minutes after the onset of the operative procedure the patient was pronounced dead.

CASE 9.—This 7-year-old boy was admitted for an elective tonsillectomy and adenoidectomy. His general physical status was excellent. The operative procedure was carried out under general anesthesia, using ether. Apparently an overdosage of the anesthetic agent was given, and acute cardiac arrest occurred. Artificial respiration was carried out immediately. Artificial circulation through a transthoracic route was instituted within four to five minutes after recognition of the absence of blood pressure and pulse. Artificial circulation for only a few minutes was followed by rapid and complete recovery of the cardiac action. The patient's postoperative course was a very complicated one. The child remained comatose and in a state of decerebrate rigidity for many weeks. He finally recovered with extensive neurologic residues and evidence of severe generalized cerebral damage.

CASE 10.—A 9-month-old boy was admitted with a diagnosis of congenital hydrocephalus and meningocele. The meningocele had previously been repaired. There was paralysis and anesthesia below the level of T 10. The lungs were recorded as normal. A cerebellar craniotomy and a Torkilsen procedure was planned. The anesthesia was begun with open drop vinethene and an oral endotracheal tube inserted. Small doses of nembutal were given intravenously for basal narcosis and nitrous oxide 70 per cent—oxygen 30 per cent were given by means of the semi-open technique. One hour and a half after the surgical procedure started the heart began to slow from a rate of 120 to 80, and suddenly became inaudible. The patient was quickly turned from the prone to the supine position and a short intercostal incision was made through the fourth left intercostal space. The heart was found to be beating at a very slow rate, estimated to be about 10 to 20 beats per minute. Artificial respiration was maintained throughout this time, and artificial circulation was begun. A rhythmic beat of 120 per minute was obtained at the end of a ten-minute resuscitation period. The heart rate rapidly climbed to 200 per minute, where it remained for the next fifty minutes. At the end of this time the heart again suddenly stopped. Resuscitation at this time was unsuccessful. There was a four-minute delay from the first loss of the heart action until the first thoracotomy was completed, and artificial circulation was instituted. Upon the second attempt a six-minute delay occurred. Artificial circulation and respiration were discontinued after twenty minutes.

CASE 11.—A 63-year-old man entered the hospital with the complaint of epigastric pain of about one and one-half years' duration. A diagnosis of a gastric ulcer along the lesser curvature was made, and a subtotal gastric resection was attempted through a thoracoabdominal incision. During the procedure the cuff of the endotracheal tube in some manner obstructed the endotracheal tube, causing a period of anoxia resulting in rapid loss of blood pressure and pulse which did not respond to artificial ventilation. Artificial circulation by the transthoracic approach was started within six minutes of recognition of the onset of the cardiac arrest. This was continued for a period of forty-five minutes. Following the administration of 2 per cent procaine, 200 mg. and 5 mg. of neosinephrine intravenously, the volume of the pulse increased, and the heart rate improved so that blood pressures of about 60/40 could be obtained. The patient never resumed spontaneous respiration, and

had to be placed in a respirator postoperatively. Death occurred twelve hours after the onset of the acute cardiac episode. Post-mortem examination revealed massive pulmonary collapse, edema, and congestion of the lungs. There was moderate generalized arteriosclerosis and cardiac hypertrophy.

CASE 12.—A 68-year-old woman was admitted with the complaint of a cough for four months and hemoptysis of five weeks' duration. Electrocardiograms showed bundle branch block and an incomplete A-V block with delayed A-V conduction. The patient was hypertensive and had cardiac enlargement. There was evidence of cardiac failure at the time of admission. An additional complaint was some urinary incontinence for which the patient was seen by the Department of Urology. The urological investigation revealed that the patient had a large left suprarenal mass displacing the left kidney downward. An exploration of the left suprarenal mass was attempted. During induction of the anesthesia, under nitrous oxide, oxygen, and cyclopropane, cardiac arrest occurred. Artificial ventilation was carried out continuously and artificial circulation through a left transthoracic approach was started three minutes after the onset of the cardiac arrest. During the opening of the patient's chest, the greatly dilated heart was incised in the left ventricle which bled profusely requiring rapid replacement of blood. The cardiac muscle was sutured. Artificial circulation was maintained at all times during the repair of the cardiac muscle. A cardiac beat started again, and for a brief time blood pressure of 180/110 could be obtained. The heatbeat then became ineffective and fibrillation occurred. Intravenous procaine, quinidine, and electric shock were used in unsuccessful attempts to reverse the fibrillation. After administration of Pronestyl intracardially there was sudden and complete asystole followed in turn by a bizarre ventricular rhythm. Later the heart again fibrillated, and no repetition of the reversal could be obtained. The artificial circulation was maintained for two and one-half hours without recovery of the patient.

CASE 13.—A 10-year-old boy was admitted because of acute bulbar poliomyelitis. The following day he became comatose. Oxygen saturation studies confirmed the impression that there was inadequate ventilation, and it was decided to place an electrophrenic respirator to provide adequate ventilation. This was done and was followed by tracheotomy. No anesthesia was required for those procedures. During the performance of the tracheotomy the patient became acutely anoxic, and cardiac arrest developed. Artificial ventilation had been carried out throughout the entire period. Artificial circulation was started within three minutes following recognition of the cardiac arrest through a transthoracic approach. Artificial circulation was carried on for five minutes until a normal cardiac rhythm was established. Following re-establishment of a regular cardiac beat, the chest was closed rapidly. The patient's general condition stayed extremely poor, and the patient remained comatose and expired twenty-four hours following the onset of the acute cardiac arrest.

CASE 14.—A 25-year-old man was admitted with complaint of headaches, malaise, and fever for five days, and weakness in his legs of twelve hours' duration. A diagnosis of acute paralytic poliomyelitis was made. The paralysis involved most of the muscles of his arms and legs and the intercostals. The patient was placed in a tank respirator. His condition became slowly worse, and five days later a tracheotomy was performed to facilitate intratracheal aspiration. The patient had numerous episodes of cyanosis which were relieved by bronchoscopic aspirations. Three or four weeks later the patient's general condition improved and he was able to leave the respirator for longer periods. Three months later after supper the patient suddenly became obstructed and was observed to be deeply cyanotic; he developed cardiac arrest. The patient was bronchoscoped after about ten minutes, the foreign material aspirated, and ventilation reestablished. Through a left thoracotomy artificial circulation was started thirty minutes after recognition of the cardiac arrest. Artificial circulation was carried on for forty minutes. During the period of artificial circulation, one cubic centimeter of 1:1000 adrenalin was given into the heart. The heart was observed to fibrillate. Injection of 1.0 Gm. of Pronestyl was followed by restoration of a normal cardiac rhythm. Blood pressures were obtainable around 120/85. The patient expired twelve hours following the acute cardiac arrest without regaining consciousness.

CASE 15.—This 34-year-old man was admitted with a diagnosis of a lung abscess in the left upper lobe. The electrocardiogram preoperatively was normal. A left upper lobectomy was done. During the procedure there was some spillage of purulent material into the right main bronchus resulting in a period of hypoxia and anoxia. It was impossible to inflate the right lung for a period of time and it was thought necessary to replace the endotracheal catheter. During this procedure, the patient's heart slowed markedly and became arrested. This was immediately noted, and artificial circulation was started within half a minute from the time of onset of the cardiac arrest. Cardiac massage was carried out for one minute with complete restitution of normal cardiac beat. The operative procedure was then carried to completion without any further difficulty. The patient's postoperative course was uneventful.

CASE 16.—A 2-year-old girl had been admitted repeatedly for skin plastic procedures. She had had no difficulty during the previous operations. On this occasion during an ether anesthesia acute cardiac arrest developed and artificial ventilation was started immediately. Twenty-two minutes following recognition of the acute cardiac arrest a left thoracotomy was made; the heart was found to be in systole. Cardiac massage was instituted and 1 per cent procaine was applied to the heart. A sum total of 10 c.c. of 1:10,000 adrenalin was given into the heart. The artificial circulation was continued for about five minutes without any evident effect. The patient did not revive.

DISCUSSION

When cardiac arrest occurs and the condition is recognized, artificial ventilation with pure oxygen must be instituted immediately. The surgeon must be informed at once of the situation and, after rapid verification that circulation has actually ceased, efforts of the surgical team must be directed toward reestablishing effective blood flow. The quickest approach by which manual compression of the heart can be effected should be used. Most of the time this will be a direct transthoracic approach. Opening of the pericardium to facilitate manual compression is of great benefit. If, however, an abdominal incision already exists, transdiaphragmatic or transabdominal manipulation and compression of the heart may be tried but quickly changed to the transthoracic approach if satisfactory evidence of adequate peripheral circulation does not appear. A palpable peripheral pulse and satisfactory blood pressures must be obtained.[5] After artificial circulation and efficient artificial ventilation have been established, time has been gained to permit additional therapy if that should be indicated. This consists of the use of blood intravenously or, in some instances when blood volume has been rapidly depleted, preferably intraarterially.[6] Procaine intravenously and topically to the heart and at times electric shock have been of value.[7, 8] Epinephrine must be used most judiciously or not at all.

In order to determine experimentally whether the oxygen and carbon dioxide transport system can be adequately maintained by artificial circulation, episodes of acute cardiac arrest were produced in a series of sixteen dogs. Overdosage of either chloroform, ethyl chloride, or cyclopropane combined with hypoxia and epinephrine was used. No drugs were used for preanesthetic medication. In six dogs femoral arterial blood pressures were directly recorded on the kymogram. Arterial blood samples were drawn from the femoral artery of the opposite leg. Carbon dioxide and oxygen tensions in the blood were determined by the Van Slyke manometric method. All dogs were intubated

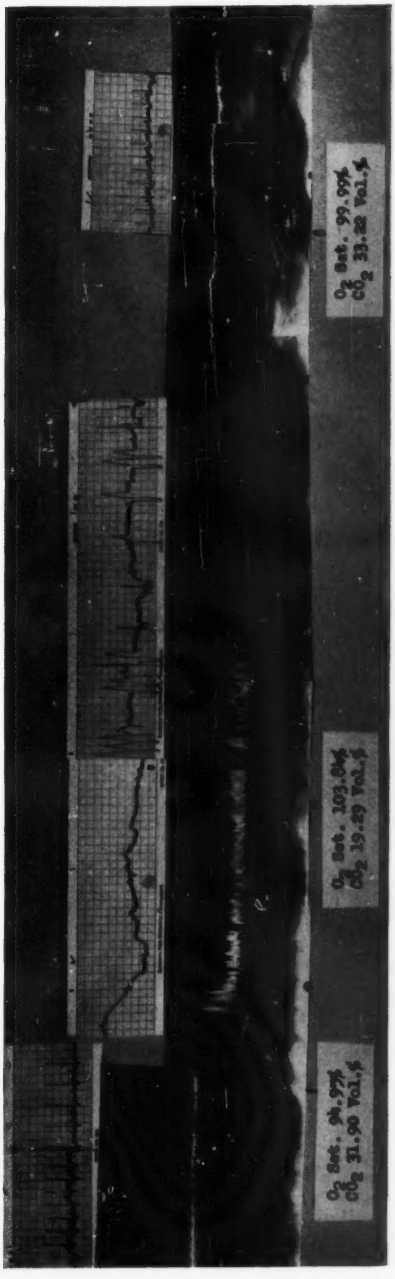

Fig. 2.—Blood pressure maintained by artificial circulation at approximately half the normal control level. Electrocardiograms show normal sinoauricular rhythm, ventricular standstill (with p waves still present), ventricular escape, and eventual return to normal rhythm.

in order to aid in adequate ventilation, and a direct writing electrocardiograph recorded continuously. Artificial circulation was instituted through a direct transthoracic approach. Thoracotomy was not carried out until cessation of effective cardiac beat was evident.

It was shown by these experiments that the mean femoral arterial blood pressure can be maintained at about approximately half of the original level. Ineptitude or fatigue of the operator resulted in distinctly lower mean arterial readings during the period of artificial circulation. Oxygen tension of the femoral arterial blood could be maintained at close to normal levels during the period of artificial circulation. Values for the arterial carbon dioxide tensions were lowered as the result of the artificial ventilation (Fig. 2).

Obviously the results obtained in experiments using mongrel dogs with apparently normal hearts cannot be compared with statistics on clinical cases. The patient with severe sclerotic[9] or congenital heart disease,[10] severe pulmonary disease, or other complicating features may not have sufficient reserve to respond even to very prompt adequate therapy. It is important to note that study of the histopathologic sections of the hearts of these patients who died following cardiac massage did not show any evidence of acute extensive myocardial damage even after prolonged manipulation. Localized petechial hemorrhages, particularly in the epicardium, as the result of the procedure of artificial circulation, were occasionally observed. The same was true of hearts obtained from the dog experiments. Similar findings have been reported by others.[11]

In summary, the following points should be kept in mind for adequate therapy. The anesthetist must be in constant close contact with the patient to be able to recognize the emergency when it arises. This is first evidenced by sudden cessation of pulse and inability to obtain blood pressure readings. Artificial ventilation with oxygen must be instituted promptly. An unobstructed airway must be present at all times, but valuable time must not be wasted by intubation of the patient until after the oxygen and carbon dioxide transport has been re-established. The time interval between recognition of the state of acute cardiac arrest and the commencement of artificial circulation should be kept to a minimum, and all persons in the room made acutely aware of the elapsed time. The rather arbitrary time limit of three to three and a half minutes may hold true under the most ideal circumstances. However, if a patient has been in a state of hypoxia for a long period of time, irreparable damage to the brain may occur in a much shorter period of additional time than that arbitrary time limit. Artificial circulation must be rapidly instituted by the surgeons, preferably through the direct transthoracic approach. Adjuvant therapy can then be instituted at leisure since the artificial circulation and ventilation will maintain the oxygen and carbon dioxide transport system. Such additional therapy may include the use of procaine intravenously, into the heart or applied topically. This may reduce the irritability of the myocardium and the possibility of ventricular fibrillation may be decreased. In the event that ventricular fibrillation does occur, artificial circulation still must be maintained. Electric shock with electrodes applied to each side of the heart should be used in an attempt to reverse the ventricular fibrillation. One very

important warning should be made. A drug which historically has been used for the treatment of cardiac arrest may have caused many deaths by producing ventricular fibrillation. This drug, epinephrine, if used, should be given in small, very dilute quantities. A dosage of 0.1 mg. in 1:10,000 dilution has been observed by us to cause ventricular fibrillation. Under no circumstances should the content of the usual epinephrine ampule be administered. If prolonged artificial circulation should be necessary, supportive measures for maintenance of the circulating blood volume are essential as intravenous or in some instances intra-arterial transfusions.[6, 12]

REFERENCES

1. Nicholson, J. C.: Cardiac Massage, Brit. M. J. **1**: 385, 1942.
2. Trent, J. C., Gaster, E.: Anesthetic Death in 54,128 Consecutive Cases, Ann. Surg. **119**: 954, 1944.
3. Gunn, J. A.: Massage of the Heart and Resuscitation, Brit. M. J. **1**: 9, 1921.
4. Barber, R. F., Madden, J. L.: Resuscitation of the Heart, Am. J. Surg. **64**: 151, 1944.
5. Dripps, R. D., Kirby, C. K., Johnson, J., Erb, W. H.: Cardiac Resuscitation, Ann. Surg. **127**: 592, 1948.
6. Johnson, J., Kirby, C. K.: An Experimental Study of Cardiac Massage, Surgery **26**: 472, 1949.
7. Beck, C. S., and Mautz, F. R.: The Control of the Heart Beat by the Surgeon, Ann. Surg. **106**: 525, 1937.
8. Beck, C. S.: Resuscitation for Cardiac Standstill and Ventricular Fibrillation Occurring During Operation, Am. J. Surg. **54**: 273, 1941.
9. Beck, C. S., Rand, H. J.: Cardiac Arrest During Anaesthesia and Surgery, J. A. M. A. **141**: 1230, 1949.
10. Cooley, B. A.: Cardiac Resuscitation During Operations for Pulmonic Stenosis, Ann. Surg. **132**: 930, 1950.
11. Ruzicka, E. R., Nicholson, M. J.: Cardiac Arrest Under Anaesthesia, J. A. M. A. **135**: 622, 1947.
12. Fauteux, M.: Cardiac Resuscitation, J. THORACIC SURG. **16**: 623, 1947.

DISCUSSION

DR. R. STARR LAMPSON, Hartford, Conn.—I wish to emphasize, also, the importance of prompt cardiac massage in this emergency. For surgeons who are familiar with the chest, or who are working in the chest, this action is almost automatic. General anesthesia is used by all surgeons and it should be part of our duty to inform those who are not familiar with operating in the chest how to handle these emergencies. I wish to emphasize what has been said about promptly opening the chest, and I would like to suggest that it is better to open the chest unnecessarily than to open it too late.

I have had cardiac arrest occur twice in the same patient in one week. I would like to present the circumstances briefly. This girl was 12 years old at the time. She had situs inversus associated with extensive bilateral bronchiectasis. The left middle lobe and basilar segments of the left lower lobe had been resected one year before. She had reentered the Hartford Hospital for resection of the right lower lobe. At operation, May 4, 1949, through a lateral approach, just as the inferior pulmonary vein was being exposed, the heart stopped in arrest. This occurred at the same time that the anesthetist was sucking to remove tracheal secretions. The first slide, which is a copy of the anesthesia chart, depicts graphically what happened. When the child went into cardiac arrest massage was immediately started, and after five minutes the heart beat normally at a rate of about 140. We decided not to proceed with the operation; it was deemed better judgment to stop and carry on another day. The child was sent back to the ward and had an uneventful convalescence from this episode. Six days later she was brought back to the operating room.

(Slide.) This slide shows a summary of the second anesthesia chart. The operation was in almost exactly the same situation, the inferior pulmonary vein had just been ligated, the anesthetist had again applied suction, and the child again went into cardiac arrest. Massage was instituted, and normal beats appeared in about four minutes. Intravenous pro-

caine was used in both instances, and the second time the procaine had been infiltrated around the hilum to try to avoid this unfortunate sequence of events. The lobectomy was completed. The child had an uneventful convalescence and was discharged from the hospital on the twelfth postoperative day, in complete possession of her mental faculties, and so far as I know her heart has beaten without interruption since.

DR. DWIGHT E. HARKEN, Boston.—Dr. Ehrenhaft's work is always thorough, reliable, and constructive. When such a study involves Dr. Cullen's magnificent department of anesthesia, it will always stand the closest scrutiny and represent a substantial contribution.

I rise, not as one who has had extensive experience with successful defibrillation of the heart or one who has inordinate confidence in the efficacy of cardiac massage, but rather as one who has had considerable experience and many unfortunate contacts with some very poorly functioning hearts on the operating table.

I am greatly impressed by one point in particular. We have used the term "cardiac arrest." This is the term that is in vogue. It seems to call for some sort of heroic action. This is not necessarily cardiac arrest. All the anesthesiologist can report is "circulatory arrest" because he obtains no blood pressure and can feel no pulse. If an electrocardiograph is running, we may find that the electrical impulses are coming through. *Circulatory arrest* may be (a) ventricular fibrillation, (b) cardiac arrest or standstill, (c) coronary occlusion, (d) pulmonary embolus, or (e) any of a number of circulatory catastrophes such as ruptured aneurysm, etc. Let us reflect briefly on the best treatment and the place for emergency thoracotomy and cardiac massage. (We will use the term "intermittent cardiac compression" to avoid confusion.) Some of these five categories of circulatory arrest can be rectified by very simple means of therapy. In others, prevention is the practical attack. Still others are disadvantageously managed by thoracotomy and intermittent cardiac compression.

If the cause of this circulatory arrest is ventricular fibrillation, how good is our defibrillation equipment and how rapidly is it available? I personally am dissatisfied with existing defibrillating equipment. The work of Claff and Dickson in our laboratory would suggest that we are not using very good defibrillating equipment unless we give one or two very brief shocks of 140 volts at 3 amperes. This is not available in most of the equipment designed for the purpose of defibrillating the heart. Until such instruments for defibrillation and reinstitution of heartbeat are available, I believe that we have to consider this aspect of the correction of circulatory arrest unsatisfactory.

When the circulatory collapse is due to or coincident with the insertion of an intratracheal tube, the insertion of a Levine tube, manipulation of the hilum, manipulation of the mesentery, the gallbladder or the stomach, I think it is fair to think that that is the thing called vago-vagal *cardiac arrest*. How much of a part anoxia plays is not altogether clear, but certainly there is a vagal factor. It can be favorably influenced prophylactically by vagal block in those patients who have it repeatedly during surgery. During operation most of these patients who have such cardiac standstill during intratracheal aspiration, etc., respond immediately and completely to a simple flick of the heart with the finger or, if the chest is not open, to a prick with the needle. Those that really need and profit most by intermittent cardiac compression are, in our experience, those patients who have had too much anesthetic agent with too little oxygen or too much retention of carbon dioxide and perhaps some underlying myocardial disease. These true arrests respond to intermittent cardiac compression. However, certainly it would be better to treat them before the fact, just as pulmonary embolus is probably best managed through prophylaxis. The analogy between this heroic surgery and the Trendelenburg operation is not far-fetched; time will, I suspect, complete it. The under-use of agents rather than the overuse (quantity and variety) by anesthesiologists will probably liberate us from this responsibility.

We need not stress the disadvantages of intermittent cardiac massage in the individual who has had a coronary occlusion. The patient with this form of circulatory arrest is likely to forego his last opportunity at survival the minute his chest is incised. This probably is not an uncommon cause of sudden death on the operating table. We need not say

more about surgical intervention for pulmonary embolus. Its best treatment again is in prevention and there are few of you who are sufficiently enthusiastic about the Trendelenburg operation to maintain those kits in the hospitals like fire extinguishers.

The position of thoracotomy and intermittent cardiac compression is not clear in my mind. I am deeply concerned about the effect of this unbridled enthusiasm in this direction on patients throughout the world. I think that all the above factors should be carefully weighed by surgeons as they formulate their own policies. It is very easy to say *"They would have died anyway."* This is not always true. Many alert anesthesiologists encounter circulatory arrest frequently. Many of these are transient. We all have a great responsibility to report our total experience so that the proper course can be plotted. We are indebted to Dr. Ehrenhaft for his excellent report of his total experience.

DR. CHARLES P. BAILEY, Wyncote, Pa.—Unlike the previous speaker, I do come here to speak as one who has had quite a lot of experience with cardiac standstill. I do not know if true cardiac arrest can occur, but with the exception of cases of ventricular fibrillation, we have seen very little evidence to suggest that a heart vigorously beating suddenly stops. It usually slows down and gets weaker and then stops. As chest surgeons we have actually seen a number of hearts do just that thing. I do not know how many cases of cardiac standstill we have dealt with, but it must be in excess of fifty during our respective careers. Eighteen of them are living and well. Two of those who are well had primary ventricular fibrillation, but not more than six of the total had primary ventricular fibrillation, which we consider a rare incident. We have found our entire success in the program of correction of this condition to be based upon two features. One is adequate training of the anesthetist in recognizing the condition. The other is prompt thoracic intervention. We have trained our anesthetists so that they no longer feel around and finally, after several minutes, say "I can't get a pulse." Now, when they suspect that the patient's condition is not good, they feel for the carotid artery, and if they do not feel a pulse they say to the surgeon "The patient is dead. His heart is stopped. Will you open the chest or shall I?" This replaces the usual fatal hesitation and doubts by a surgical reflex, and the incision is made.

As a result, gynecologists, urologists, and even an eye surgeon at our institution have now explored the heart with the proper thoracic incision, and have saved cases of standstill during such operations as circumcision, prostatic resection, and cataract extraction. We do not think three and a half minutes is an accurate time to set for these emergency thoracotomies. The chest should be opened at once. We have seen irreparable cerebral damage with only one minute of visualized standstill, and we have had recovery in our series in a patient without heartbeat for eight minutes. That means without massage, either. She had no subsequent evidence of cerebral damage. I was called in to see one patient who was said to be sixteen minutes without heartbeat, who subsequently recovered with but minimal neurologic signs.

We do not fear the use of adrenalin, and we use it for two purposes; one is to restore myocardial tonus if we have a case of fibrillation, ventricular fibrillation that will not respond to electric shock. We give adrenalin in doses ranging up to 5 c.c. That is usually the third administration. We give 0.5 c.c. the first time. That improves the heart muscle tone and then they may come out of fibrillation with proper electric shock. The other purpose is to improve the heartbeat after overcoming the standstill. Adrenalin will pep up a weakly contracting myocardium which seems about to stop as soon as massage is stopped. In such cases a rapid strong beat can often be established with adrenalin. You can give as much as needed, but try 0.5 c.c. the first time.

As to defibrillation, I am very happy to hear what Dr. Harken said because it parallels our experience; 110 volts at 5 to 10 amperes is the minimum current that will defibrillate an adult ventricle in our experience. We are not talking about dogs. They may require only 30 or 40 volts, but human beings need at least 110 volts. If it is a very large heart even that will not stop it. In giving electric shock, therefore, we have gradually abandoned all rheostats. We now insert an ordinary plug into the wall socket or into an electrical

extension. We have two metal contacts, two oval pieces of molded metal with bakelite handles. One is put under the heart within the pericardium, and one is placed on top after you have compressed the myocardium rhythmically for several minutes and think you have it in pretty good condition. A nurse inserts the plug, usually momentarily the first time, then pulls it out. But if that fails, we have it plugged in while we count, 1-2-3; and if that is not enough we have them plug it in and count to 10 and then pull it out. That, which I know is a lethal current, will usually defibrillate the average adult human heart. But a heart cannot be defibrillated with less than a lethal current. In a few large hearts we have had to use our 220 volt line.

DR. J. H. KAY, Baltimore.—The following comments are based upon experimental and clinical observations of cardiac arrest. Cardiac massage is one of the most important tools in the treatment of cardiac arrest. The pericardium should be opened; the heart should be squeezed firmly 35 to 45 times per minute. In our experience many cases of cardiac arrest in the form of ventricular standstill have begun to beat with cardiac massage alone. Many other cases responded to cardiac massage plus injection of various cardiac stimulants.

In the past there have been two methods recommended for the treatment of cardiac arrest in the form of ventricular fibrillation: (1) the injection of procaine associated with massage; and (2) massage with electrical defibrillation. Experimentally, we have injected procaine into hearts which are fibrillating, and massaged them for periods up to eight minutes, and in not one case were we able to defibrillate the heart by this method. Apparently the only consistently successful method of defibrillating the heart is by electrical means. The heart should be massaged until it is no longer dilated or cyanotic; this usually requires one to two minutes. A 130 volt shock lasting one second or less is then passed through the heart and this will frequently defibrillate a human heart in ventricular fibrillation. This voltage may be obtained by using a transformer. If the heart is not defibrillated on the first attempt, it should be massaged for another minute or two and a series of shocks, each lasting one-third of a second, with one-third second interval between shocks, should be passed through the heart. If a series of six to eight shocks is unsuccessful, the heart should again be massaged and the series of shocks repeated. If many series of shocks fail to defibrillate the heart, 5 c.c. of 1 per cent procaine should be injected into the left ventricular cavity, the heart massaged for a minute, and electrical defibrillation again attempted. It is best not to use procaine unless absolutely necessary for it makes the ventricular standstill that may follow electrical defibrillation more difficult to treat. Following successful defibrillation, the heart may beat or it may be in standstill. If it is in standstill, it should be treated by massage and the injection of a cardiac stimulant if necessary.

We have experimented with four cardiac stimulants: isopropylepinephrine, barium chloride, calcium chloride and adrenalin. The isopropylepinephrine and barium chloride were not satisfactory stimulants. Calcium chloride and adrenalin were very satisfactory stimulants. The dose of calcium chloride for human beings is 2 to 4 c.c. of a 10 per cent solution. We differ with the other men who have spoken this afternoon as to the dose of adrenalin. We feel the dose of adrenalin should be much smaller, $\frac{1}{10}$ to $\frac{1}{3}$ c.c. of 1:1000. We feel it is important to use this small dose because a larger dose may produce ventricular fibrillation and it is very difficult to defibrillate a heart in which the fibrillation is the result of the injection of adrenalin.

The substances should be injected into the left ventricular cavity, for after a few massages they are in the coronary vessels and the myocardial bed.

DR. JULIAN JOHNSON, Philadelphia.—I think there are a couple of other points that might be mentioned. First, I think that we, as thoracic surgeons, owe a certain obligation in all the clinics to spread this information to general surgeons. I hope we have succeeded in doing that in our own institution at the University of Pennsylvania in the last five years, where we have performed approximately 1,000 operations per month, and have attempted to resuscitate the heart in twenty instances. Only nine of those, however, were actually in the operating room at the time; two were undergoing cardiac catheterization

and nine were in the ward. During the first three years we were successful in only one of four attempts; during the last two years we have been successful in seven out of seven attempts; that is, for those who were in the operating room or its equivalent. I make that point because our anesthetists now notify us quickly. We have taught the general surgeons, residents, and the like, to open the chest quickly. In only four of the eleven instances were they thoracic patients; the others were general surgical patients of one sort or another.

Our experience with ventricular fibrillation has been rather different from some others. In half our patients who have had arrest, fibrillation occurred at one time or another. It has been our experience that no heart has remained arrested, that we have either been able to get it back to normal rhythm or it has gone on into ventricular fibrillation on its way to demise. We have felt, however, that if we have got the patients sufficiently early so that we could get them oxygenated again, we have been able to defibrillate them by electric shock. Of the ten, we were able to defibrillate eight. Four of those have survived and are apparently well.

I think it might be worth while to say that if you are building a defibrillator, it would perhaps be well to put an isolating transformer on it. The current as it comes out of the wall has one hot wire and one ground wire. If you have a hole in your glove, with the hot wire you may get an electric shock. On the other hand, with the isolating transformer there is no hot wire. You cannot get a direct current from one wire even though you are grounded yourself. It would be necessary for you to have a hole in both gloves in order to get a shock when you have the transformer. We have taken the added precaution of wearing two pairs of gloves. One would have to have a hole in all four gloves, and they would have to be superimposed in order to get a shock. Your anesthetist also might wear gloves. We have given a mild shock to a couple of anesthetists who did not have on gloves.

DR. WILLIS J. POTTS, Chicago.—First, I want to congratulate Dr. Ehrenhaft on his courageous presentation. I am not sure that the following comments are entirely appropriate, if cardiac arrest is defined as the unexpected cessation of cardiac activity without obvious cause such as the anoxia of severe pulmonary stenosis. We have had one questionable case of cardiac arrest in approximately 500 cases of congenital heart disease operated upon; children between ten days and 16 years of age. Why this good fortune? I wish I knew, because each week I am worrying that the percentages may catch up with us. Our surgery is no different from that in a hundred other hospitals. If our good fortune is not due to coincidence and is not due to the fact that we are dealing only with children (as I think it is not), it must be due to some other factor and that, I believe, is anesthesia. We try to keep anesthesia extremely simple. About 95 per cent of our anesthetics have been given by Dr. McQuiston.

We give liberal doses of morphine balanced with atropine and scopolamine, because we think it avoids cardiac arrhythmia. We use only a bit of ether and cyclopropane, no pentothal, no barbiturates, rarely curare. Controlled respiration is used continuously, and in view of Dr. Beecher's comments on Monday, maybe that is of more importance than we realize.

If the child's heart rate slows the operation is stopped immediately and the lung is re-expanded until the child is oxygenated and the heart comes back to regular rhythm. About the only drug used during operation is 1/1000 grain of atropine given intravenously to quicken the heart action. However, there are no drugs in the pharmocopeia which can be substituted for oxygen.

We use the first plane of anesthesia. Often the child will squeeze the anesthetist's finger during the operation. Every anesthetic agent is a poison. The less used, the less the danger of damaging brain and cardiac function. We cool all the children who are severely cyanotic, to about 92° to 95° F. to conserve the consumption of oxygen. Usual cases such as patent ducti are kept at normal temperatures.

In summary, we emphasize and reiterate again and again that the stomach must be empty; oxygen must continuously be introduced into the lungs and the carbon dioxide must be removed; few drugs are used and a minimum of anesthetic agent. In view of the work

of many investigators, I believe, that inadequate exchange of gases and excessive anesthesia are the cause of idiopathic cardiac arrest. Solomon published an article about 4000 years ago; he said "Pride goeth before destruction and an haughty spirit before a fall."

DR. RICHARD H. OVERHOLT, Brookline, Mass.—Dr. Potts brought up most of the points I wished to mention; namely, minimum anesthesia and elimination of all drugs which may blanket out reflex mechanisms which are valuable indications for the surgeon and the anesthetist to go by. Thoracic surgeons are fortunate in having most of these accidents happen when the chest is open or when the patient is in the operating room.

Whenever the blood pressure makes a drop, we make it a practice to stop operating, take the retractor out, and release all traction on the lung. When there is any evidence of a pre-shock level, the whole team should devote its full attention to finding an explanation for why the pressure has become depressed. It is our feeling that most of these catastrophes develop early in the operation because of obstructed airways, or late in the operation because of failure to replace sufficient amounts of blood. I agree with Dr. Bailey that there are signs which give an indication of when standstill is imminent. We do not hesitate to massage the heart when it becomes slow or when the pressure is low. Many more hearts are massaged when they are still beating than after they have stopped. I think that most of these catastrophes in the thoracic patient can be avoided.

DR. EHRENHAFT (closing).—I want to thank all the different discussers, and I am sorry that I am unable to answer all the discussers individually due to lack of time. If one reviews the histories on those patients one cannot help but get the impression that most of those accidents are due to anoxia and all the other names, as, for instance, vagal reflex, given as the cause of acute cardiac arrest, fall under the same heading. It is practically always anoxia. This may be due to overdosage of the anesthetic agent, obstruction of the airway by faulty catheters, spillage of secretions to the opposite lung, and other happenings of this sort. We also would like to emphasize that the patients presented are not only patients on the surgical service; they represent patients from all services at the University Hospitals. We have tried to educate the physicians on the different services and the anesthetist since 1947 to recognize this condition, and have emphasized the points of immediate artificial circulation and adequate artificial ventilation by high oxygen concentrations. We have felt that the only really adequate way to maintain artificial circulation is by direct approach to the heart through the chest.

CARDIOSTATHORRHAPHY—SPLINTING THE FLACCID MYOCARDIUM OF CORONARY OCCLUSION BY SURGICAL PROCEDURES

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THE purpose of this experimental and clinical study was to develop a new type of operative procedure for the relief of the effects of coronary occlusion in the human heart. Experiments were carried out on the heart of the living dog with a view of improving the function of that ventricle which had been rendered less efficient by having had a portion of its wall thrown out of function by the coronary occlusion. We have devised operative procedures for splinting the flaccid, noncontractile, expanded area of the myocardium which develops after sudden occlusion of a major coronary vessel. It is probable that such operative procedures might be of benefit to the human heart which has suffered a coronary occlusion.

SECTION I

RATIONALE FOR ATTEMPTS AT SURGICAL RELIEF IN PATIENTS WITH CORONARY OCCLUSION

Surgical procedures aimed at correction of the effects of coronary occlusion in the human heart would be possible, since at least 80 per cent of patients with coronary occlusion survive for a month and even for years after the first attack. There is thus a sufficient time interval for recovery from the immediate effects of coronary occlusion to permit surgical intervention in the great majority of these patients.

There is an ever increasing amount of evidence to indicate that the pathologic human heart will tolerate operative procedures upon the heart itself. Rehn[1] demonstrated in 1896 that the human heart would tolerate operative procedures performed upon the muscle wall of the ventricle. He was the first to suture a wound in the living human heart. Graham and I,[2] in 1922, confirmed these findings and we showed that the auricles would tolerate operative procedures better than the walls of the ventricles. Cutler and Levine,[3] in 1923, proved that the pathologic myocardium of the left ventricle in the human heart would tolerate rather extensive surgical procedures carried out upon it. A careful study of the various types of surgical procedures which have been performed upon human hearts after coronary occlusion in them and which will be reviewed here indicate that such hearts will tolerate operations directly upon the myocardium itself.

The operative procedures which we shall describe and which have been devised as the result of our studies in the experimental laboratory involve the

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ischemic noncontractile area of the myocardium in the surgical manipulations. Similar nonfunctioning areas of the heart would be involved in the surgical manipulations of the heart in patients who have had coronary occlusion. One would expect these nonfunctioning fibrotic areas of the myocardium to tolerate surgical procedures even better than the functioning portions of the myocardium.

THE EFFECTS OF CORONARY OCCLUSION IN THE MYOCARDIUM

It has been known for the past fifty years experimentally that the ischemic zone of the myocardium distal to a coronary artery which has been ligated and thereby occluded becomes flaccid. Baumgarten,[9] in 1899, was among the first investigators to note that the ischemic zone of the myocardium distal to the occlusion bulged outward during systole. Twenty years before this however, in 1881, Cohnheim and von Schulthness-Rechberg[7] noticed that the ischemic zone became first pale and then bluish in color. Long before this, according to Mallory, Chirac[6] in 1698 had proved that the coronary artery was essential for maintaining the life of the myocardium. He tied off a principal branch of the coronary artery in the heart of the dog and noted that it stopped beating almost immediately.

Karsner and Dwyer[10] studied the histologic changes in the infareted area of the myocardium distal to a coronary occlusion by ligation in the heart of the dog. These investigators found first a hyaline necrosis of the muscle fibers, and this was followed by the formation of collagen and then connective tissue which gradually replaced the necrotic myocardial cells. The necrotic muscle was carried off by the phagocytic monocytes, and at the end of eighteen days it had been replaced by a well-defined scar.

Histologic studies made on human hearts with coronary occlusion show a progression of the processes of healing similar to that described by Karsner and Dwyer in the heart of the dog. Levine,[13] in 1929, in his monograph on "Coronary Thrombosis; Its Various Clinical Features," found the necrotic muscle of the myocardium to be replaced by connective tissue which was demonstrable on the sixth or seventh day. Mallory, White, and Salcedo-Salgar[5] studied the speed with which healing takes place in the infarcted area of the myocardium distal to a coronary occlusion. Signs of removal of the necrotic myocardial muscle fibers appeared during the first week after the occlusion. During the second week removal of the peripheral muscle fibers by the phagocytes is a prominent histologic feature. During the third week the first signs of collagen formations are found in the form of fine collagen fibrils which are produced by the fibroblasts at the periphery of the infarct. The amount of collagen increases during the fourth to the sixth weeks, the connective tissue becomes more dense during the sixth and seventh weeks, and at the end of two months has reached its maximum. The infarct has been replaced with connective tissue at the end of eight weeks, and can be considered to be practically healed.

Tennant and Wiggers[12] found that almost immediately after tying off a branch of the coronary artery of the heart of a dog the ischemic area ceased

to contract, and reversal of movement took place. The area stretched during systole. The amount of this stretching was measured by mechanical means, by the myocardiograph.

Sir Thomas Lewis[11] observed that the ischemic area became darker in color shortly after ligation of the anterior descending branch of the left coronary artery. In this observation he confirmed the previous observation of Cohnheim and von Schulthness-Rechberg. In some of his animals fibrillation developed, and the animals died. In other experiments the heart continued to beat, though less efficiently, owing to part of its wall being thrown out of action. He stated, "In the human subject similar events undoubtedly happen."

THE LOCAL EFFECTS OF CORONARY OCCLUSION IN THE HUMAN HEART

Sir Thomas Lewis' observation that the ischemic zone of the myocardium distal to a coronary occlusion in the human heart might bulge with each systole has been abundantly proved. It is often possible to see the abnormal movement of the ischemic zone of the myocardium through the fluoroscope, but this abnormal movement is better recorded by the roentgenkymogram.

Roentgenkymography is an accurate method of recording the movement of several points on the surface of the myocardium on a single x-ray film. This permits the comparative analysis of the movements of the different portions of the wall of the heart, and by careful measurements one can determine whether the infarcted area distal to an occlusion develops a paradoxical movement or whether it moves synchronously with the remainder of the myocardium or whether its movement is diminished. It has become well known clinically that the ischemic portion of the myocardium may bulge or balloon out during systole of the ventricle.

Stumpf,[14] Scott and Moore,[15] Hirsch,[16, 17] Gubner and Crawford,[18] and Bourdet and Fishgold[19] were among the first ones to demonstrate by roentgenkymography that the ischemic segment of the myocardium distal to a coronary occlusion often develops a paradoxical movement when compared to the movement of the remaining mass of the myocardium. The ischemic or infarcted zone of the myocardium moves outward during systole instead of contracting inward as does the remainder of the myocardium.

A most significant study for our consideration here is that reported by Sussman, Dack, and Master[20] and the companion article by Dack, Sussman, and Master[21] on "The Roentgenkymogram in Myocardial Infarction." These investigators studied two hundred cases of typical coronary occlusion at the Coronary Occlusion Clinic of the Mount Sinai Hospital in New York City. Eighty-five of these cases were those of recent coronary occlusion occurring within the two months before the time of reporting. In the remaining 115 cases the lesion was classified as being old. The roentgenkymogram showed abnormalities of the infarcted area in 75 per cent of the two hundred cases. These abnormal changes were an expansion of the ischemic infarcted area during all or a part of systole or a diminished movement of the infarcted area.

It is of interest also that these changes in the roentgenkymograph following coronary occlusion remained for a long period of time. Sixteen of twenty-two

patients who were examined from four to twelve years after their initial attack still showed abnormalities in the roentgenkymogram which were classified as complete or partial systolic expansion.

Dack, Sussman, and Masters[21] made a correlation between the degree of recovery of the patient and the presence of complete systolic expansion of the infarcted area, and in those patients with marked diminution of pulsation of the infarcted area and the degree of recovery. In those patients with complete systolic expansion of the infarcted area, 57 per cent were classified as having poor recoveries, and in those patients with marked diminution of pulsation of the infarcted area only 13 per cent were classified as having poor recovery.

It would seem quite probable that those hearts which showed complete systolic expansion of the infarcted area were those in which the scar tissue of healing had not contracted down as a firm cicatrix, perhaps because of its size. Those in which the infarcted area showed minimum movement would seem to be those in which healing had been satisfactorily accomplished.

When viewed in the light of our experimental work to be reported here, the unfavorable prognosis of those patients in whom the infarcted area showed expansion during systole assumes a great significance. Our experimental work indicates that it would be possible to change an area which was expanding during systole into an area which would not expand and, hence, would correspond to the spontaneous, satisfactory healing with firm stiff scar formation. If we could convert an infarcted area of the myocardium which showed systolic expansion of the area to one which showed a marked diminution of the movement of the area by splinting and compressing it by surgical means, we would expect to bring about clinical improvement and a better prognosis for the patient.

Section II

Revascularization of the Myocardium

The term revascularization of the myocardium was used by Claude Beck to designate the attempt to increase the blood supply to the myocardium through the establishment of anastomotic channels between the systemic circulation and the coronary circulation. Thorel[22] and Moritz, Hudson, and Orgain[27] were among the first to observe anastomotic channels between the systemic and coronary circulations. These anastomoses were in pericardial adhesions. To Beck,[23] however, goes the honor of having proved that blood actually passes along these anastomotic vessels during life. He cut such an adhesion at operation in November, 1934, and he noticed active bleeding occurring from each end of the transected tissue. The bleeding appeared to be more brisk from the cardiac end than from the pericardial end.

We might properly ask ourselves whether it would be possible to increase the blood supply to the myocardium in a pathologic heart. Normally the myocardium is abundantly supplied with capillaries. In fact it is difficult for us to conceive the richness of the capillary supply to the myocardium. Wearn[33] injected the capillary system of the myocardium with India ink and found by actual count that each myocardial fiber is in direct contact with one or more capillaries. The ratio between the myocardial fibers and the capillaries in the

adult human heart is one to one. Wearn found an average of 5,738 capillaries per cubic millimeter of the myocardium in the left ventricle of the human heart. This is the equivalent of 94,027,000 capillaries per cubic inch of this portion of the myocardium. The ratio of one capillary to one myocardial fiber remains constant throughout life, and it remains constant even in pathologic hearts. In arteriosclerosis of the coronary artery the capillaries are not involved in the pathologic process, only the larger vessels of the coronary arterial tree are involved. In a successful revascularization of the myocardium, therefore, it would be necessary to replace only the larger vessels by new anastomotic channels.

EXPERIMENTAL REVASCULARIZATION OF THE MYOCARDIUM

Beck and Tichy[23] roughened up the epicardium and the interior of the pericardium of the hearts of dogs, and allowed these tissues to grow together by adhesions. Silver clamps were applied to the right coronary artery, to the descending branch, and the circumflex branch of the left coronary artery. These clamps were closed with varying degrees of completeness to produce partial obstruction or complete obstruction of the branches of the coronary arteries. They found when the coronary arteries were obstructed that new vessels grew into the myocardium from the systemic circulation.

In another series of experiments, Beck and Marko[24] removed the epicardium and occluded the major coronary arteries in stages. Almost the entire coronary tree could be occluded in this manner with survival of the animal. Beck concluded that the presence of such a collateral circulation in the human heart might protect the patient from subsequent sudden occlusions of a major coronary artery.

Beck used the pericardium and the voluntary muscles of dogs or the fatty tissue as the source of the systemic collateral circulation through the myocardium. These tissues were sutured to the myocardium. Heinbecker and Barton[29, 30] felt that it should be unnecessary to suture muscle tissue or the pericardium to the myocardium, and they therefore injected foreign irritants into the pericardium to produce the adhesive pericarditis. They introduced powdered aleuronat and lionite, which is more commonly known as carborundum, into the pericardium of dogs. The extent of the anastomosis between the coronary system and the systemic circulation following such procedures was astonishing. At post-mortem when the systemic circulation was injected, so much fluid ran through the anastomotic channels into the myocardial vessels that it was necessary to make an opening into the cavity of the left ventricle to allow the fluid to escape from it. They tested the efficiency of the collateral circulation in their dogs by tying off both the anterior descending branch of the left coronary artery and at the same time the circumflex branch of the left coronary artery, a procedure which is nearly always immediately fatal. Of fourteen dogs in which both the anterior descending and circumflex branches of the left coronary artery were ligated simultaneously, eight died immediately, but six lived from one to three months afterward.

REVASCULARIZATION OF THE HUMAN HEART AFTER CORONARY OCCLUSION

The knowledge which has been gained in the experimental laboratory in the production of a collateral circulation between the systemic circulation and the coronary circulation has led to operations on the human heart which has suffered a coronary occlusion. Beck[23] in 1936 reported having operated upon eleven patients, six of whom were living and in whom the results were good or acceptable. The pectoralis muscles were used most often but the intercostal muscles, the subcutaneous fat, the mediastinal fat, and the triangularis sterni muscles were used also. In the twelve years between 1936 and 1948 Beck[25] reported an additional twenty-one patients in whom a collateral circulation had been produced by operative means. The results of these operations were sufficiently satisfactory to make it seem worth while to continue this type of operative procedure.

Thompson and Raizbeck[4] introduced talcum powder into the pericardial sac under direct vision, through an incision, into the pericardium. Twenty-seven of thirty-six patients were observed from the time of the operation up to as long as nine years after the operation or until the time of death, and 70 per cent of them were definitely improved. These patients were severely incapacitated, and the authors therefore consider the results of this form of surgical rehabilitation of the conorary cripple as being excellent.

O'Shaughnessey[32] brought up the omentum in experimental animals and in the human being to produce a collateral circulation between the vessels in it and the coronary arteries. He, too, felt that his results were encouraging.

Beck[31] made an actual anastomosis between the systemic arterial tree and the coronary sinus. In the dog he anastomosed the common carotid artery with the coronary sinus, and in his first patient he used a free graft of the brachial artery to establish the anastomosis between the side of the thoracic aorta and the coronary sinus. In this operation a reversed flow of the blood is established, since the coronary sinus is the terminal vessel of the great cardiac veins.

One can hardly imagine a more spectacular or a more difficult operation than this operation of anastomosis of the systemic arterial tree to the coronary sinus. What operation could offer a greater challenge than that of sewing an artery to the coronary sinus of the living pulsating heart! It has been difficult, however, to determine the soundness of this operation from a physiologic viewpoint. Beck found that it might be possible to supply too much blood to the myocardium through such an anastomosis, and it is probable that the high blood pressure in the venous system of the myocardium would not be altogether desirable. He concluded that beneficial effects followed such an operative procedure in the hearts of dogs and that the degree of improvement was sufficient to be regarded as significant.

SECTION III
OUR EXPERIMENTS ON REGIONAL REVASCULARIZATION OF THE MYOCARDIUM

Most of these operative procedures directed toward increasing the blood supply of the myocardium have contemplated the formation of an extensive adhesive pericarditis. Heinbecker and Barton point out that this sterile

adhesive pericarditis is different from the constrictive pericarditis which develops spontaneously in the human heart. It seemed to us, however, that it might be more desirable to establish these adhesions only between that part of the myocardium which was supplied by a particular branch of the coronary artery and which had become occluded.

Clinically significant infarction of the myocardium in the human heart is found but rarely in the right ventricle. The clinically significant infarction involves the wall of the left ventricle. It would seem logical, therefore, that an increase to the blood supply of the myocardium of the left ventricle would be sufficient in the human heart. Accordingly, we planned three series of experiments aimed at increasing the blood supply to the left ventricle and at the same time to leave the right ventricle uninvolved in the adhesive pericarditis. In the first two series adhesions were established between the pericardium and the myocardium of the left ventricle; in the third series of experiments we attempted to increase the blood supply to certain areas of the myocardium of the left ventricle through the development of a collateral circulation between branches of the left coronary artery itself.

1. In the first series of these experiments adhesions between the pericardium and the myocardium of the left ventricle were produced by roughening up the epicardium of the entire left ventricle and its corresponding area of the pericardium. This roughening up process was carried out first by the use of cloth-backed sandpaper. It was not possible always to confine the pericardial adhesion to the left ventricle when we used sandpaper because particles of sand would become dislodged, and through the motion of the heart would become lodged on the right ventricle and would produce adhesions there. When we substituted a perforated metal thimble for the sandpaper the adhesions formed between the left ventricle and its pericardium alone when we were careful to remove the blood from the pericardial sac at the end of the operation. Seventeen dogs were used in this series of experiments. We found that it was possible to establish a collateral circulation between the vessels in the pericardium and the coronary arteries of the left ventricle.

2. In our experiments we noticed that adhesions formed along the suture line in the pericardium. We therefore carried out a second series of experiments in which a number of parallel sutures were implanted through the pericardium overlying the left ventricle. These sutures were placed from within the pericardial sac and they outlined the boundaries of the left ventricle. Adhesions formed between the pericardium and the myocardium of the left ventricle and through these adhesions a collateral circulation was established between the systemic circulation in the pericardium and the coronary circulation within the walls of the left ventricle.

3. In our third series of experiments designed to increase the blood supply to the myocardium of the left ventricle, sutures were placed in the wall of the left ventricle. It is well known that a hyperemia develops around a foreign body. We felt that strips of fascia or strips of the pericardium when drawn through the myocardium would act as a foreign body and would produce a greater hyperemia than would silk sutures. Accordingly, strips of pericardium one-eighth of an inch in width were left attached near the conus arteriosus and

then threaded on a needle and drawn through the myocardium of the left ventricle alongside and parallel to the anterior descending coronary artery.

Microscopic examination of the myocardial tissue immediately surrounding the strip of pericardium showed congestion of the capillaries as a rule, but in

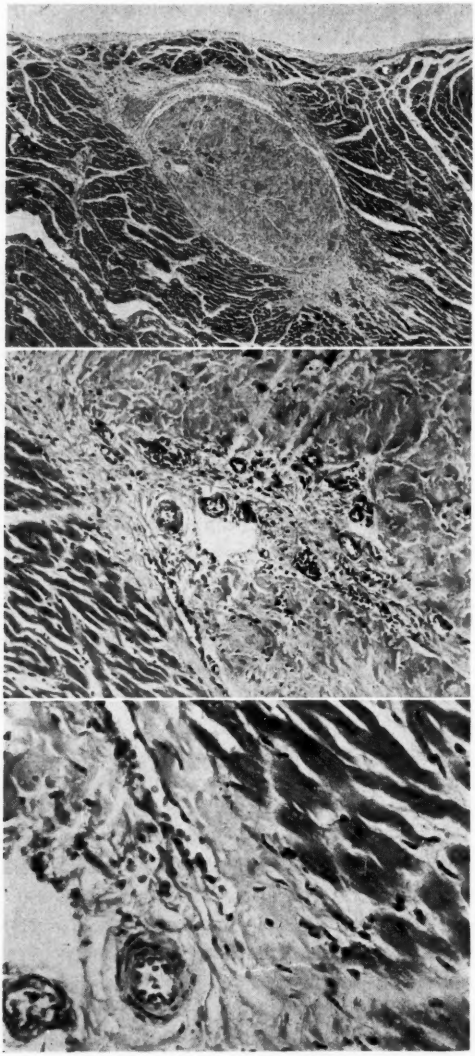

Fig. 1.—Microphotographs of a cross section of a strip of pericardium which has been embedded in the myocardium. The section is taken at a level where the embedded strip is near the epicardium. The low-power magnification shows the absence of inflammatory reaction at the end of three weeks.

B, The area outline on *A*, ×130. Note the absence of necrosis in the adjacent myocardium and the presence of four fairly large arteries between the myocardium and the embedded strip of pericardium. *C*, A higher magnification of two of these arteries (×340). These arteries contain red cells.

one of these experiments there were large vessels in the vicinity of a strip of pericardium (Fig. 1). These vessels are larger than one would expect to find normally in the myocardium at this depth. We were not able to prove to our own satisfaction whether these vessels had been carried into the depths of the myocardium along with the strip of pericardium or whether they were abnormally dilated branches of the coronary arterial tree. They contained normal red blood cells and seemed, therefore, to be functioning vessels.

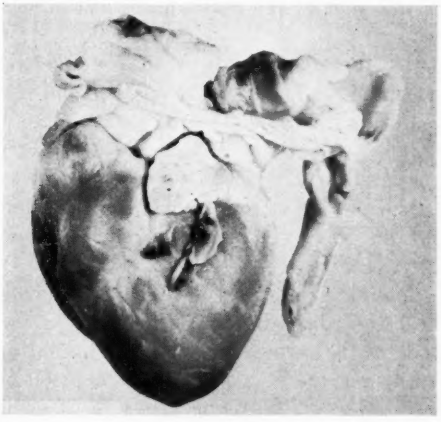

Fig. 2.—Photograph of heart of experiment 49 HB-33, removed seven days postoperatively. The pericardium has been reflected from the heart. It was adherent to the anterior wall of the left ventricle in the region of the ischemia. It was gently stripped off for inspection of the myocardium.

Note the two silk suture-ligatures around the large branches of the anterior descending coronary artery. Note also the loop of the pericardial strip suture and the normal appearance of the myocardium in the zone of ischemia.

Section IV

Compression and Splinting of the Ischemic Area of the Myocardium

In our experiments designed to revascularize a specific area of the ischemic myocardium described in the previous section of this paper, a strip of pericardium was drawn through the ischemic area of the myocardium which had resulted from ligation of branches of the anterior descending branch of the left coronary artery. The strip of pericardium was embedded so that its course ran parallel to the course of this vessel. This strip was left attached to the pericardium near the conus arteriosus. Its free end was then attached by a single stitch to the apex of the left ventricle. Thus in these previously described experiments this strip of pericardium lay loosely stretched in its channel through the myocardium.

THE CRUCIAL EXPERIMENT

In experiment 49 HP-33, May 17, 1949, we ligated only two primary branches of the anterior descending coronary artery. A zone of cyanosis appeared in the myocardium of the left ventricle along the course of these arteries. This zone was about three-fourths of an inch in width and one and one-half inches in length.

An unusually long strip of pericardium had been fashioned and threaded on the needle, and had been brought through this zone of cyanosis. Instead of attaching the free end of the strip of pericardium to the myocardium near the apex of the left ventricle as we had done in previous experiments it was looped back and sewn to itself, as shown in Fig. 2. As we did this we noticed that the cyanosis had disappeared from the zone of the myocardium which had been supplied by the two vessels which had been ligated. The myocardium again assumed a color quite like that of the surrounding tissue which had not had its blood supply lessened!

We had noticed that the distance between the ligated blood vessels was greater after ligation than it had been before ligation, and that there was bulging of the ischemic area. This indicated that the tonus of the myocardial fibers in the ischemic zone was less after ligation than it was before. After tightening up of the strip of pericardium these ligated vessels were again drawn toward each other. Thus the size of the ischemic area of myocardium had been reduced back to its original width. We had established slight compression of the ischemic area.

We were both fascinated and perplexed by the change in color of the ischemic area of the myocardium in this experiment. It seemed clearly to be due to the compression of the myocardial tissue by the suture. Was the change in color the result of squeezing out the excess stagnant blood from the ischemic area or did the compression of the tissue bring about an improvement in the efficiency of the circulation?

It has been firmly established that compression of certain tissues of the body helps to bring about healing in them, for example, it has become commonly known that a pressure bandage on an indolent ulcer of the leg will be beneficial for healing of the ulcer if the pressure bandage is maintained for a long enough period of time. Perhaps the explanation for this is in elimination of the edema. In plastic surgery skin grafts, skin flaps and other transplanted tissues are more likely to survive if they are compressed slightly with a pressure bandage. It seemed probable to us that compression of the infarcted area of the myocardium following occlusion of its blood supply might indeed be beneficial in bringing about healing of the ischemic area. The pressure exerted by the sutures on the ischemic myocardium produces an effect similar to the pressure bandage on other tissues.

Besides these beneficial effects resulting from compression of the ischemic area there is the beneficial effect of stabilization of the flaccid and stretched ischemic zone of the myocardium. The ischemic area became stabilized after the ligature-strip of pericardium had been tightened and it no longer bulged during systole.

A NEW TYPE OF OPERATIVE PROCEDURE

These observations made on the heart of this experimental animal gave rise to the development of a plan for a distinctly different operative approach for the treatment of the ischemic area of the myocardium which follows coronary occlusion in the human being. Almost all previous attempts by ourselves and by

others had been directed toward revascularization or toward the production of an increase in the blood supply to the myocardium. This had been done either through the establishment of a collateral circulation from the systemic arteries or from an attempt to increase the blood supply to the ischemic area from the coronary arterial system through anastomotic channels. Our new operative procedure was not intended to establish a collateral circulation to the ischemic area. It is directed toward the mechanical fixation of the oscillating, flaccid, ischemic area of the myocardium distal to an occlusion of the coronary artery and to compressing this area back to as nearly its original normal size as possible.

There are several different operative procedures which will bring about stabilization of the ischemic area, and at the same time will compress the area back to its normal size. We have done this by fixing the ends of strips of pericardium to the myocardium at the edge of the ischemic area and then bringing the strip of pericardium outside the epicardium to attach it to the heart at the opposite side of the ischemic area. These sutures are placed in such a manner that when they are tightened the ischemic area will be stabilized and compressed back to its original size. In this technique the strips of pericardium do not run through the ischemic area of the myocardium. They run over its external surface.

A second operative procedure which we have used and in which strips of pericardium are bridged over the surface of the heart to produce compression and stabilization is to fashion wide strips of pericardium and by interrupted silk sutures to attach the edge of the strip to the myocardium along the border of the ischemic area. A second wide strip is fastened in like manner to the opposite border of the area. When these two strips of pericardium are joined together with interrupted silk sutures the ischemic area is compressed and at the same time it is prevented from bulging during systole.

In still a third type of operative procedure the pericardium was allowed to become adherent to the ischemic area, and at a second operation strips of fascia lata were woven through the adherent pericardium in such a manner as to stabilize and compress the ischemic area. In others a sheet of fascia lata was attached to the adherent pericardium at its edges and then the sheet of fascia lata was plicated in a manner similar to that used by Beck[35] in his operation for aneurism of the human heart.

Our operation for stabilization of the ischemic area of the heart differs distinctly from that of Beck, however, since Beck's operation was designed to prevent rupture of the aneurism and our operative procedure is designed to stabilize and diminish the area of the flaccid, ischemic myocardium or the flaccid sheet of fibrous tissue which replaces it. Beck described his operation very nicely in setting forth his purpose, "The operation consists of grafting a segment of fascia lata, or parietal pericardium, over the aneurism for the purpose of preventing its rupture." The object of all of our operative procedures has been to compress and to stabilize the area of the myocardium distal to an occlusion of the coronary artery.

It seemed to us that less disturbance in the blood flow through the larger branches of the coronary artery followed placing of the strips of living suture

material through the myocardium of the ischemic area, than when we allowed these strips to pass over the surface of the ischemic area. We shall therefore limit the discussion of this operative procedure to those experiments in which the suture material was placed through the myocardium of the ischemic area and in which the same suture was brought back through the myocardium to form a loop.

RATIONALE FOR SPLINTING THE ISCHEMIC AREA OF THE MYOCARDIUM

It is well known clinically and experimentally that an ischemic portion of the myocardium may bulge or balloon out during systole. Sir Thomas Lewis,[11] as we mentioned before in this paper, found that if a branch of the coronary artery such as the anterior descending branch is tied in a dog an area of the ventricular wall loses its blood supply in part. It becomes darker in color and bulges with each systole of the heart. If fibrillation does not follow, the heart continues to beat, though less efficiently, owing to part of its wall being thrown out of action. The changes in color may be seen readily by the naked eye, and with careful observation the ballooning may be seen to occur during systole if the heart rate is not too rapid.

Recently Prinzmetal and his co-workers[36] have studied the onset of ballooning during systole immediately after ligating the anterior descending branch of the left coronary artery in anesthetized dogs. These investigators made slow motion moving pictures of the heart before, during, and after ligation of the arteries. They found slight ballooning appearing about fifteen seconds after the artery was tied. The ballooning often waxes and wanes, and sometimes disappears. It may disappear and return again. Especially is this true in the left ventricle. In some of their experiments the ballooning gradually became more intense until it occupied the whole of systole. If the area of ischemia is large enough, ballooning persists.

Prinzmetal and his co-workers increased the work of the ventricle in the dog after ligation of the anterior descending coronary artery by placing a large clamp on the aorta and closing it partially to raise the blood pressure to a higher level. They found that when ballooning was not present before this procedure it occurred during the increased work of the heart, and if ballooning was present before clamping the aorta it was greatly intensified during the acute hypertension. This suggests that increased work on the damaged heart of the patient with coronary occlusion has a deleterious effect.

These investigators then studied patients who had chest pain after exercise. They found that in some instances the systolic ballooning of the ischemic area of the myocardium occurred after exercise which was not present before exercise. They suggest that since the ischemic myocardium balloons more with exercise and since clinical angina pectoris is usually brought on by exercise, that possibly the spontaneous intermittency of ballooning is related to the spontaneous intermittency of pain clinically.

There is therefore clinical and experimental evidence that when a large branch of the coronary artery is occluded suddenly the area of the myocardium

which receives its blood supply from the occluded artery loses its tonus, becomes flaccid, and a paradoxical movement develops in the ischemic area. It bulges outward during systole when it should be contracting inward, and since the tonus of the myocardial fibers is less the whole area expands out to form a zone larger than it was before the occlusion. This puts added work upon the remainder of the muscle mass of the affected ventricle.

ANATOMIC CONSIDERATIONS

In order to understand the embarrassment to the efficiency of the affected ventricle which stems from the presence of the flaccid, oscillating and noncontractile area of the myocardium it is well to consider the anatomic structure of the muscles which make up the heart.

According to Morris' Anatomy,[37] the ventricular musculature is very complex and consists of numerous groups of muscle fibers distinguished from one another by their direction. In general, the fibers of the deepest layers have a direction crossing those upon the surface of the same area at an angle. The intervening layers of fibers have all stages of obliquity.

All fibers arise from and are inserted into the fibrous partitions at the base of the ventricles. Their origins and their insertions are either directly into the trigona or into the fibrous bands of tissue surrounding the mitral orifice or the orifice of the tricuspid valve or into the membranous septum between the ventricles. Some of these bands are inserted indirectly through the chordae tendineae and the valves. These bands of myocardial fibers form a somewhat continuous muscular sling which originates at the base of the ventricle and whirls around the heart, sometimes twice around the left ventricle, to return to the same base of the heart and to be inserted there.

When the myocardial fibers which make up one of these bands contract, the band as a whole shortens, thereby squeezing the walls of the ventricle closer to each other in systole. If one would sever this band in its middle portion, it would be logical to expect that its efficiency would be greatly decreased. If one would sever all of these bands of myocardial tissue near their middle, the heart would indeed become a very inefficient pump and perhaps could not propel the blood forward even if the epicardium were intact.

A physiologic severance of many of these bands takes place following occlusion of the anterior descending branch of the left coronary artery or the occlusion of a sufficient number of its principal branches.

If we consider the action of one of these bands of myocardial fibers in which a portion of the band had ceased to contract and had become stretched and flaccid, we can see that the remainder of the band of muscle would be obliged to contract more violently to overcome the handicap of this nonfunctioning link of muscle tissue. It would seem logical, therefore, that if we could decrease the handicap arising from the stretched zone of myocardial tissue by tightening up its elongated fibers, the remaining functioning myocardial fibers of the band could perform more efficiently and with less effort.

INCOMPLETE EMPTYING OF THE VENTRICLE

There is a second load placed upon the remaining functioning muscle fibers of the myocardium in addition to that arising from the lack of teamwork of the fibers in the ischemic area of the heart. This additional load derives from the fact that the ischemic area bulges outward during systole.

A part of the contents of the ventricle is shot into the ballooned ischemic area during systole instead of being propelled forward through the aorta or through the pulmonary artery. In some of our experiments we have attempted to measure this increased amount of work by the left ventricle following ligation of the descending branch of the left coronary artery. We measured the amount of blood propelled by the left ventricle before and after ligation of the coronary artery. Using a rotometer, we attempted to determine whether or not there was a decrease in the total flow of blood through the heart after ligating several branches of the anterior descending coronary artery. We were not successful in detecting a change in the total flow after ligation of two or three or four of these principal branches, perhaps because of the inadequacy of apparatus. That there is an improvement in the stroke volume of the heart after stabilization of the flaccid ischemic area seems very probable, since Gordon Murray[39] found an astonishing increase in stroke volume after resection of the ischemic area. He suggested the possibility of resection of the flaccid expanded ischemic area in patients with coronary occlusion.

THE RESERVE POWER OF THE HEART

It is of interest and importance here to consider briefly the reserve power of the heart. Prinzmetal and his group,[38] in one of their careful studies on the coronary circulation, investigated the problem. They call attention to the fact that animals can live with 25 per cent of one kidney, 40 per cent of one lung, 20 per cent of the liver, 16 per cent of the adrenal cortex, and 6 per cent of the intestines. These figures have been arrived at by removal of a portion of the intestine or of the kidney or of the liver in animals. It is not practical to remove fractions of the myocardium, but it is possible to throw out of function graded portions of the myocardium, especially of the left ventricle, by graded ligation of the branches of the coronary artery supplying it.

These investigators made slow-motion moving pictures of the living heart as they were tying off the arteries supplying the left ventricle. As the blood supply was decreased to an area supplied by the ligated vessels, this area ballooned out and as the next branch of the left coronary artery was tied the corresponding area of the myocardium ballooned out also. They tied the large branches of the left coronary artery beginning near the apex, and proceeded slowly upward to avoid fibrillation. Their experiments showed that the resting blood pressure of the animal could be maintained by only 15 per cent of the myocardium of the left ventricle.

Comment.—It is to be noted that these experiments were carried out on the normal hearts of dogs under anesthesia and at rest. There must be some question as to whether these figures and these results could be translated over to the

pathologic human heart. They do show, however, the remarkable reserve power of the heart of the dog, and it is probable that the human heart has a similar great reserve power although it is likely that the human heart could not be expected to carry on over the years with only 15 per cent of its muscle mass being able to function.

In patients with coronary occlusion there is seldom more than 10 or 15 per cent of the myocardium thrown out of function in those patients who survive the immediate effects of the occlusion. In view of the great reserve power of the heart, it seems possible that the detrimental effects of coronary occlusion might be due to a paradoxical movement of a portion of the wall of the left ventricle as well as to a loss of function of this area of the myocardium. If so, a surgical procedure which would splint this nonfunctioning area of myocardium and which would thereby prevent paradoxical movement of it might render the heart a more efficient pump. If this could be accomplished, a crippled heart might be rendered more efficient in the human being with clinical coronary occlusion.

A third series of experiments was devised to test whether a zone of nonfunctioning myocardium might be splinted by strips of pericardium or strips of fascia or other strong tissues woven through the ischemic area and anchored so as to prevent paradoxical movement of the area.

EXPERIMENTAL PROCEDURES IN SPLINTING THE ISCHEMIC AREA OF MYOCARDIUM

Dogs were used in these experiments. They were given nembutal anesthesia intravenously and placed on their right side. An intratracheal tube was introduced and connected to an intermittent positive pressure tank so as to produce an intermittent type of artificial respiration. The sixth rib was removed subperiosteally, the pleural cavity opened, and the lung packed aside.

The pericardium was then opened along the course of the anterior descending branch of the left coronary artery. This descending branch of the left coronary artery was ligated near its middle portion or in other experiments two, three or four of the principal branches of the descending anterior coronary artery were ligated with silk. Strips of pericardium or of fascia lata about ⅛ inch in width were then woven through the ischemic area from one of its edges to the other. These suture materials were placed as mattress sutures bridging through the myocardium from side to side across the narrowest width of the ischemic area. They were placed so as to avoid those larger vessels which could be seen through the epicardium. Their course was laid close to the endocardial surface of the left ventricle but they did not penetrate into the cavity of the left ventricle. A special needle was devised to carry the strips of pericardium or other living suture material. It has a hollow shank in which the end of the strip of living suture material can be placed and this is anchored to the needle by a fine strand of silk. The needle therefore does not produce as much trauma as it would if the end of the strip of suture material were passed through an eye.

As these sutures are being placed care is exercised not to tie them too tightly nor at the same time to tie them too loosely. In the acute experiment we

tried to produce enough pressure by the suture material to reduce or to obliterate the cyanosis of the ischemic area and to eliminate ballooning of the area (Fig. 3).

The pericardium was then closed and the lung carefully re-expanded to fill the pleural cavity. The chest wall was closed in layers and the dog was allowed to recover.

Comment.—During the operation the heart was observed carefully for changes in color following ligation of the anterior descending artery or its branches, and the changes in color of the ischemic area after the splinting sutures had been placed. It was possible in the majority of our experiments to bring the ischemic portion of the myocardium back toward the normal color.

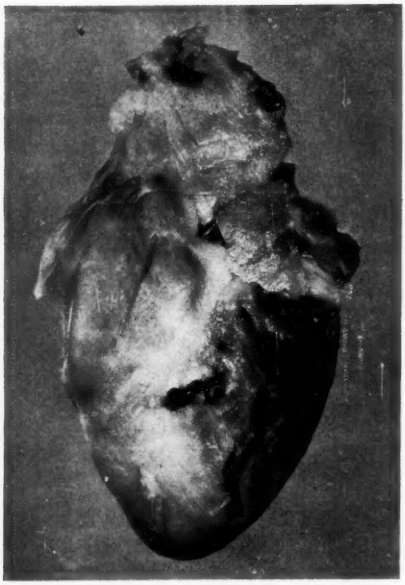

Fig. 3.—Three of the principal branches from the anterior descending coronary artery have been ligated and the zone of ischemia has been compressed slightly with a mattress suture. There was no ballooning of the ischemic area after the mattress suture had been placed and tightened.

It was always possible to bring the ischemic area back to its normal size or to tighten up the sutures to compress it even smaller than the normal whenever we wished to do so.

POSTOPERATIVE OBSERVATION

We were concerned in the postoperative period to notice whether or not any of our experimental animals developed ventricular fibrillation or whether they developed a change in rhythm of the heart. None of the hearts exhibited such changes in rhythm and none of them developed fibrillation so far as we could determine.

After the immediate postoperative recovery period these animals went about their cages or their runways as though no operative procedure had been carried out upon the heart. They showed no evidence of pain in the region of the heart.

POSTOPERATIVE AND POST-MORTEM EXAMINATION OF THESE HEARTS

These experimental animals again were given nembutal intravenously sufficient to produce anesthesia at seven, fourteen, twenty-one, or twenty-eight days. The operative wound was again reopened, the lung detached from the costal pleura and if it were adherent to the pericardium it was also stripped off that structure. The pericardium was found to be adherent to the heart along the line of suture in most of these experiments. The heart and the pericardium were removed en bloc without opening the pericardium. The pericardium was then split at a distance from the operative field and it was lifted up and the heart and the inner surface of the pericardium examined. In most of the experiments there were no adhesions between the pericardium and myocardium except along the line of suture of the pericardium and at the point where the ligature had been placed around the coronary arteries or in the field of the ischemic area.

We were particularly careful to note whether or not the splinting sutures had worn down into the myocardium. We had thought that with the continuous movement of the beating heart the ends of these suture loops might produce a pressure atrophy since they were tied with tension. There was no gross evidence of this having occurred to any considerable degree in these short-term experiments.

The microscopic examination of the myocardium showed a surprisingly small amount of reaction around the strips of pericardium or the strips of fascia. Fig. 1 is typical of the microscopic picture at the end of three weeks. The myocardium had grown to the strip of pericardium.

RESULTS IN THESE EXPERIMENTS

The results which we wish to record here of fixation of the ischemic area of the myocardium are those which we could observe in the acute experiment and the pathologic findings in the ischemic area of the left ventricle at the end of one, two, three and four weeks.

In these experiments it was possible to produce ballooning of the ischemic area if a sufficiently large number of the principal branches of the descending coronary arteries were ligated. It was also possible to produce ballooning of the ventricle if we ligated the anterior descending branch itself at or above its midportion. We could correct this ballooning by placing our sutures through the ischemic area in such manner as to bring the ischemic area back to its normal size or when we compressed it into a width less than its normal size.

After ligating the anterior descending branch of the left coronary artery or of two, or three or four of its principal branches a cyanotic area develops in the myocardium distal to the obstruction of the artery. We could cause this darker discoloration to disappear in whole or in part by compressing the ischemic area with sutures.

These compression sutures were placed immediately after the production of the obstruction in the coronary artery. It would not be possible today to place the compression sutures in the myocardium of the ischemic area after spontaneous occlusion had occurred in a coronary artery of the human heart but our observations in the normal heart of the dog suggest that if they could be placed in the human heart immediately after coronary occlusion the results might be beneficial rather than detrimental.

The improvement in the quality of the completeness of contraction of the left ventricle following placing of the tension-stabilizing sutures was often sufficient to be detectable by observing the heart with the naked eye. In two of our experiments after ligation of the anterior descending branch of the left coronary,

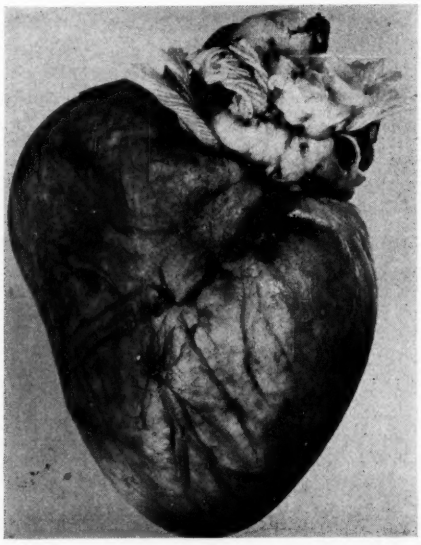

Fig. 4.—The aorta was clamped off and the living left ventricle filled with India ink. The ischemic area is sharply defined. India ink is seen in the ischemic area but in much less quantity than in the nonischemic area. Compare the width of the ischemic area in Fig. 4 with the width of the ischemic area in Fig. 5.

there was sufficient dilatation of the entire heart to produce cyanosis of the entire myocardium. Shortly after the tension-stabilizing sutures were placed and tightened up the color of the heart became more nearly normal and the quality of the contractions of the left ventricle improved. There was no doubt in our minds at the time that the heart had become more efficient after stabilization and compression of the ischemic area.

At autopsy one, two, three, or four weeks after coronary occlusion, produced by tying off the descending anterior branch of the left coronary artery or its principal branches, there was no bulging of the ischemic area in any of the experiments.

Gross examination of the hearts of these twenty-one experiments showed necrotic areas of considerable size in three of the experiments. These necrotic areas were near the endocardial surface of the myocardium. In control animals a line of demarkation between the ischemic area and the normal myocardium could be seen (Fig. 4). This is especially well shown in those hearts in which the coronary arteries were injected with India ink. The ischemic areas in the control animals were much lighter in color than the surrounding normal myocardium. In those hearts in which the ischemic area had been splinted and reduced back to its normal size before injection of the India ink into the coronary arteries, the difference in color in the two areas was not so marked (Fig. 5).

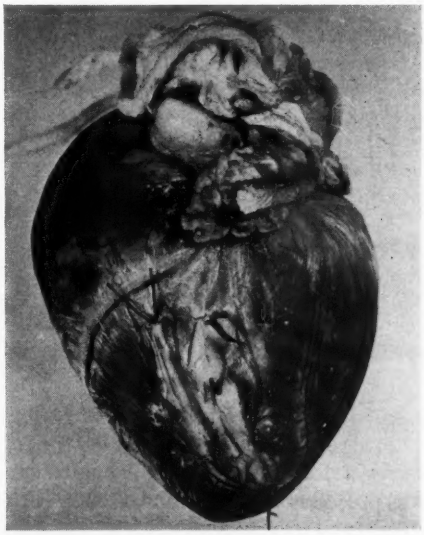

Fig. 5.—India ink has entered the myocardium through the coronary arteries, the dying heart acting as its own pump. The ischemic area has been compressed with two mattress loop sutures. Compare the width of the ischemic area in Fig. 5 with the width of the ischemic area in Fig. 4.

RÉSUMÉ

These experimental studies and clinical considerations furnish evidence that the ischemic area of the myocardium distal to a coronary occlusion loses its tonus and develops a paradoxical movement. In both the heart of the experimental animal and in the human heart with coronary occlusion these changes in the ischemic area of the myocardium come on shortly after the coronary vessels have become occluded. In the human heart this ballooning is sometimes progressive in type and it is increased after exercise in certain cases. This increase in size of the ischemic area is accompanied at times by the onset of the pain of angina pectoris.

We might ask ourselves what is the cause of the deterioration of the heart after occlusion in one of the larger branches of the coronary artery. Is it due

to the fact that the ischemic area of heart muscle ceases to do its share of work and thus more work is thrown upon the remaining functioning myocardial fibers; is it due to the presence in the heart wall of a flaccid, stretched, noncontractile link in the band of the myocardium; or is it due to progressive arteriosclerotic changes in the larger vessels of the coronary arterial system? It is probable that all three of these factors play a part in bringing about a decrease in the efficiency of the heart and this decrease is a progressive one over the long term.

In the past fifteen years a great deal of experimental work has been done toward revascularization of the myocardium in an effort to counteract the effects of a subsequent coronary occlusion by having previously established a collateral circulation between the systemic arterial system and the coronary arterial system. In the experimental animals this collateral circulation has been established to the degree that it is possible to ligate both the anterior descending coronary artery and the circumflex branch of the left coronary artery at the same time with survival of the animal. Operations aimed at forming such a collateral circulation have been performed on patients who have had coronary occlusion. The results in these cases make it appear that this type of operation is worth while.

Arteriosclerosis in the coronary arteries is almost always present in hearts with coronary occlusion. Up to the present time it has not been possible to prevent the occurrence of arteriosclerosis in the coronary vessels nor to arrest its progress. The effects of the ever decreasing blood supply to any of the tissues of the body as the result of arteriosclerosis is well known. Progressive damage to the myocardium in these cases with coronary occlusion is due in part to this cause.

Revascularization of the myocardium, which is really supervascularization of the myocardium, apparently could counteract some of the effects of arteriosclerosis in the coronary arteries, but it would not be expected to change the course of the sclerotic processes in the coronary arteries themselves. It is encouraging to remember that arteriosclerosis in the coronary system affects only the larger vessels and it does not extend down into the capillaries of the myocardium. Thus the capillary vessels are maintained at a more nearly normal state and we need only to solve the problem of preventing arteriosclerosis in the larger coronary arteries.

The total amount of work required of each ventricle of the heart is determined by the demands of the tissues of the body for blood. Normally this work is carried on by the entire musculature of the ventricle following coronary occlusion. If 10 per cent of the muscle fibers cease to contract permanently then the remaining 90 per cent of the myocardium must carry on the same amount of work. There is therefore an added load thrown upon the functioning myocardial fibers following coronary occlusion. The deleterious effects of this added load upon the functioning myocardial fibers is at once evident.

Revascularization of the ischemic area of the myocardium through establishment of a collateral circulation into it from the systemic circulation might lead to resumption of function of these myocardial fibers if atrophy had not

progressed too far in them. I have not been able to find experiments in which revascularization of the myocardium had been produced after atrophy of the ischemic area had been allowed to develop.

The ischemic area of the myocardium becomes flaccid and spreads out and a paradoxical movement develops in it. Since the heart is a pump this flaccid, noncooperative zone in its wall is detrimental to it. This is a mechanical defect and it can be remedied by a surgical procedure.

We have described here the principles of a new type of operative procedure, namely, the splinting and stabilizing of the ischemic area of the myocardium distal to a coronary occlusion. In addition the ischemic area of the myocardium is reduced back to its normal size.

In the experimental animal we have been able to do this by the use of sutures made from strips of pericardium or strips of fascia. These strips of living tissues have been used as sutures and they have been woven through the myocardium of the ischemic area. In other experiments the edges of sheets of fascia lata have been sutured to the myocardium around the border of the ischemic zone, or to the adherent pericardium over the ischemic zone. The sheets of fascia lata were then drawn taut to stabilize and to compress the ischemic zone. The improvement which can be seen in the function of the heart of the dog following this type of operation is sufficient to indicate that such an operative procedure would be of benefit to the human heart which had suffered a coronary occlusion.

I should like to express my thanks to Dr. Evarts A. Graham, Professor of Surgery, Washington University School of Medicine, who gave encouragement in this experimental work, and to Duff Allen, Jr., who made valuable suggestions and who assisted at operation in many of these experiments.

REFERENCES

1. Rehn, I.: Ueber Penetrierende Herzwunden und Herznach, Arch. f. Klin. Chir. **55**: 315, 1897.
2. Allen, Duff S., and Graham, Evarts A.: Intracardiac Surgery; A New Method, J. A. M. A. **79**: 1028, 1922.
3. Cutler, E. C., and Levine, S. A.: Cardiotomy and Valvulotomy for Mitral Stenosis, Boston M. & S. J. **188**: 1023, 1923.
4. Thompson, S. A., and Raisbeck, M. J.: Surgical Rehabilitation of the Coronary Cripple, Ann. Int. Med. **31**: 1010, 1949.
5. Mallory, G. Kenneth, White, Paul D., and Salcedo-Salgar, Jorge: The Speed of Healing of Myocardial Infarction, Am. Heart J. **18**: 647, 1939.
6. Chirac, P.: De Mortu Cordis, Adversaria Analytica, p. 121, 1698.
7. Cohnheim, J., and von Schulthness-Rechberg: Ueber die Folgen der Kranzarterienverschliessung fur das Herz, Virchows Arch. f. Path. Anat. **85**: 503, 1881.
8. Kolster, R.: Experimentelle Beitrage zur Kenntnisse der Myomalacia Cordis, Skandinavi Arch. f. Physiol. **4**: 1, 1893.
9. Baumgarten, W.: Infarction of the Heart, Am. J. Physiol. **2**: 243, 1899.
10. Karsner, H. T., and Dwyer, J. E.: Studies in Infarction. IV. Experimental Bland Infarction of the Myocardium, Myocardial Regeneration and Cicatrization, J. M. Research, **34**: 21, 1916.
11. Lewis, Sir Thomas: Diseases of the Heart, New York, 1933, The Macmillan Co., p. 43.
12. Tennant, R., and Wiggers, C. J.: Effect of Coronary Occlusion on Myocardial Contraction, Am. J. Physiol. **112**: 351, 1935.
13. Levine, S. A.: Coronary Thrombosis; Its Various Clinical Features, Medicine **8**: 245, 1929.
14. Stumpf, P., Weber, H. H., and Weltz, G. A.: Roentgenkymograpische Bewegungslehre innerer Organe, Leipzig, 1936, Georg Thieme.

15. Scott, Wendell, and Moore, Sherwood: Roentgenkymography in Diseases of the Heart, J. A. M. A. **107**: 1951, 1936.
16. Hirsch, I. S.: The Recording of Cardiac Movement and Sounds by Roentgen Ray—(Kymophonoroentgenography), Radiology **22**: 403-422, 1934; Radiology **23**: 720-757, 1935.
17. Hirsch, I. S., and Gubner, R.: Application of Roentgenkymography to the Study of Normal and Abnormal Physiology, Am. Heart J. **22**: 413, 1936.
18. Gubner, Richard, and Crawford, J. Hamilton: Roentgenkymographic Studies of Myocardial Infarction, Am. Heart J. **18**: 8, 1935.
19. Bordet, E., and Fishgold, H.: Verification de la loi de Starling par la Radiologie du coeur, Arch. de Mal. du Coeur **28**: 198, 1935.
20. Sussman, Marcy L., Dack, Simon, and Master, Arthur M.: The Roentgenkymogram in Myocardial Infarction. I. The Abnormalities in Left Ventricular Contraction, Am. Heart J. **19**: 453, 1940.
21. Dack, Simon, Sussman, Marcy L., and Master, Arthur M.: The Roentgenkymogram in Myocardial Infarction. II. Clinical and Electrocardiographic Correlation, Am. Heart J. **19**: 464, 1940.
22. Thorel, C. H.: Pathologie der Kreislauforgane, Ergebn. allg. Path. und path. Anat. **9**: 559, 1903.
23. Beck, Claude S., and Tichy, V. L.: The Production of a Collateral Circulation to the Heart, Am. Heart J. **10**: 849, 1934-1936.
24. Beck, Claude S., and Marko, H. E.: Venous Stasis in the Coronary Circulation, Am. Heart J. **21**: 767, 1941.
25. Beck, Claude S.: Revascularization of the Heart, Ann. Surg. **128**: 843, 1948.
26. Hudson, Chas. L., Moritz, Alan R., and Wearn, Joseph T.: The Extracardial Anastomoses of the Coronary Arteries, J. Exper. Med. **56**: 919, 1932.
27. Mortiz, A. R., Hudson, C. L., and Orgain, E. S.: Augmentation of the Extracardiac Anastomoses of the Coronary Arteries Through Pericardial Adhesions, J. Exper. Med. **56**: 927, 1932.
28. Brooks, Barney, and Martin, Kirby: Simultaneous Ligation of Vein and Artery, J. A. M. A. **80**: 1678, 1923.
29. Heinbecker, Peter, and Barton, W. A.: Operation for Development of Collateral Circulation to the Heart, J. THORACIC SURG. **9**: 431, 1940.
30. Heinbecker, Peter, and Barton, W. A.: Effective Method for Development of a Collateral Circulation to the Myocardium, Ann. Surg. **114**: 186, 1941.
31. Beck, Claude S.: Revascularization of the Heart, Ann. Surg. **128**: 854, 1948.
32. O'Shaunessey, L.: Surgical Treatment of Cardiac Ischemia, Lancet **1**: 37, 1935.
33. Wearn, Joseph T.: The Extent of the Capillary Bed of the Heart, J. Exper. Med. **47**: 237, 1928.
34. Roberts, J. T., Wearn, Joseph T., and Boten, Irene: Quantitative Changes in the Capillary-Muscle Relationship in Human Hearts During Normal Growth and Hypertrophy, Am. Heart J. **21**: 617, 1941.
35. Beck, Claude S.: Operation for Aneurysm of the Heart. Ann. Surg. **120**: 34, 1944.
36. Prinzmetal, Myron, Schwartz, L. L., Corday, E., Spitzler, R., Bergman, H. C., and Kruger, H.E.: Loss of Myocardial Contractility After Coronary Artery Occlusion, Ann. Int. Med. **31**: 429, 1949.
37. Morris, Henry: Morris' Human Anatomy, edited by J. Parsons Shaeffer, ed. 10, Philadelphia, 1942, The Blakiston Company, p. 594.
38. Corday, E., Spitzler, R., and Prinzmetal, Myron: The Remarkable Reserve Power of the Heart, Ann. Int. Med. **31**: 450, 1949.
39. Murray, Gordon: The Pathophysiology of the Cause of Death from Coronary Thrombosis, Ann. Surg. **126**: 523, 1947.

THE USE OF THE AURICULAR APPENDAGE AS AN AUTOGENOUS MYOCARDIAL GRAFT

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As a result of recent developments in heart surgery, the auricular appendage has been proved to be a resectable structure.[1] While in the past grafts of somatic muscle have been used as transplants on the myocardium, the feasibility of autografts derived from the auricular appendage has not been investigated.[2] In the present experiments free grafts of heart muscle have been transplanted on the normal or surgically altered hearts of dogs, and the regenerative capacity of the involved tissues were studied after a fixed lapse of time.

A total of eighteen dogs divided into three groups were used. In the animals of Group 1, the auricular appendage was excised and transplanted upon the ventricular myocardium of the same animal. In Group 2 dogs, the auricular appendage was grafted over a segment of ventricle which was rendered ischemic by ligation of a peripheral branch of the coronary artery. In the animals of Group 3, a full thickness segment of ventricle was excised and the wound closed with the interposition of the myocardial graft. Bilateral resection and autografts of both auricular appendages were performed on two dogs from each group.

All the operative procedures were done on normal, adult mongrel dogs. The animals were anesthetized with sodium pentobarbital intravenously, intubated, and maintained throughout the operations under positive pressure respiration. The thorax was entered through the fourth intercostal space by an anterolateral incision. The left lung was packed away from the heart with a moist pad, and the pericardium was opened below the course of the phrenic nerve. A 2 per cent concentration of procaine was instilled into the pericardial cavity bathing the heart in the solution. The tip of the auricular appendage was grasped with a clamp, and the base secured with a curved rubber shod hemostat. After undersewing with interrupted mattress sutures, the auricular appendage was removed. On three occasions, after resection, a reinforcing suture was required for complete hemostasis. The excised appendage was cut along its folded border in order to form a flat muscular sheet. It was then wrapped in saline gauze to make it ready for transplantation. In the Group 1 series, the graft was placed over and fixed to the normal ventricle on its anterior surface by interrupted silk sutures. Prior to fixation, the epicardial surface of the ventricle and the endocardial surface of the auricular appendage were mildly scarified. In the dogs of the second group, a branch of the anterior

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descending coronary artery was ligated, and the graft was fixed over the area of devascularization. In the third group of dogs, a section of the ventricle was excised between a row of through-and-through sutures. The graft was placed in the ventricular defect and tied with the aforementioned sutures. In none of the operations was the pericardium resutured. Antibiotics were placed in the thoracic cavity. The thoracic wall was closed without drainage after the lung was re-expanded.

During the first postoperative week the animals were injected daily with penicillin and streptomycin. At the end of ten weeks the surviving animals were sacrificed and examined. The size of the graft was determined at operation and compared with the size found at necropsy. Sections were taken through the graft site for microscopic examination.

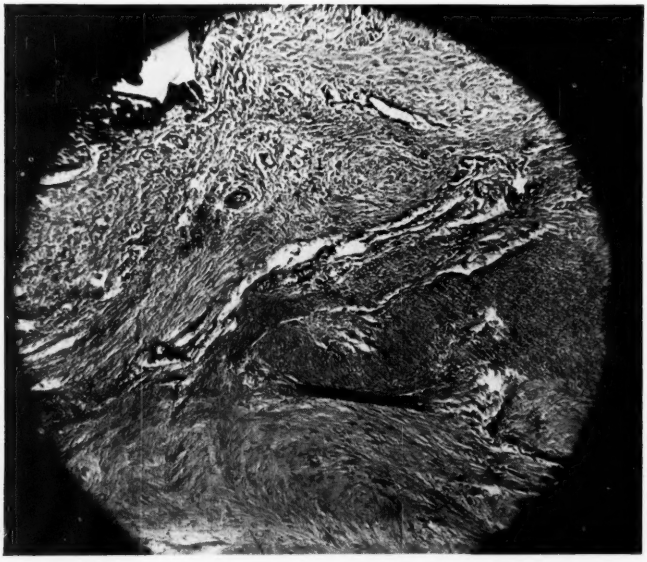

Fig. 1.—(Dog No. 4.) The auricular appendage was grafted over a segment of normal ventricle. Firm and contiguous union can be seen between the graft and the ventricular surfaces. In the upper pole a remnant of silk suture is visible.

The results of the operative procedures and other data have been summarized.

Group 1 (Free Myocardial Graft on Normal Ventricle).—All six dogs survived both the operation and the ten-week postoperative period. Each graft was found to be firmly attached to the ventricle but was reduced to an average of approximately one-half to one-fourth its original size. Pleura, pericardium, and lung were consistently found adherent to the transplant. There were no clinical manifestations of impairment of cardiac function during the postoperative life of any of these animals. Except for the changes that involved

the experimental procedure, the heart on gross examination was normal with no evidence of hypertrophy or degeneration. The site of amputation of the auricular appendage was well healed. There were no compensatory changes involving the auricle from which its appendage was removed.

Group 2 (Free Myocardial Graft over a Zone of Apparent Anoxemia of Ventricular Myocardium).—Five dogs survived the experimental procedure, while one dog succumbed due to pyopericardium on the third postoperative day. In one of the surviving dogs which had a similar infection as the latter, the transplant was absorbed. In the remaining four dogs, the graft was firmly adherent to the ventricle but was reduced in size as in the previous group. Adhesions were principally of the pleuropericardial type. The hearts were normal in size and showed no evidence of pathologic change except for the moderate scarring induced by ligation of the coronary artery branch.

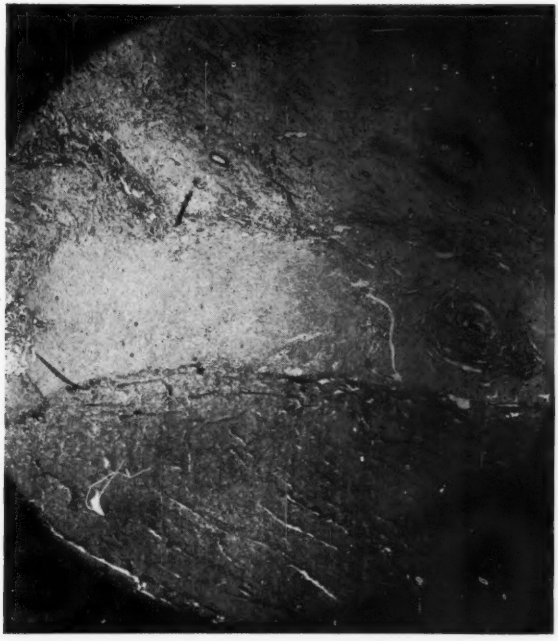

Fig. 2.—(Dog No. 8.) The auricular appendage was placed over a segment of ventricle which was first rendered ischemic by ligation of a peripheral branch of the coronary artery. In the center of the photograph the scarred ventricle can be seen. Above it is the graft and to the right there is evidence of union of the myograft and ventricle.

Group 3 (Free Myocardial Graft in Ventricular Wound).—Three dogs completed the entire experimental period. Two of the other dogs died early in the postoperative period due to hemorrhage. The remaining dog died on the sixth day from hemorrhage due to the slough of a controlling ventricular suture. The same types of adhesions as previously described were observed. The grafts

were firm and secure between the edges of the ventricle, but showed the same diminution in size. There were no compensatory or pathologic changes in the remainder of the hearts.

Microscopic examination of the tissue made through the transplant site revealed a characteristic histologic pattern in all three types of experiments. The auricular graft could be identified because of the difference in staining quality; the ventricular myocardium took on a deep red stain, while the former appeared pale pink in contrast. Union between the two muscular tissues was firm and contiguous without any distinguishing boundary between the two except for an occasional area of mild inflammatory reaction or a border of limited fibroplasia or an accumulation of myoblasts. The donor blasts were well vas-

Fig. 3.—(Dog No. 14.) Photograph shows section through graft site involving the interposition of auricular appendage muscle. Ventricle musculature can be recognized on the left side extending to the center, the lighter appearing tissue is the graft and along the right side ventricle muscle can be identified again.

cularized and showed numerous small blood vessels and other endothelial lined structures. There was a similar type vascularization in the contiguous ventricular tissue, however, the vascular structures were not as numerous. In the regions where the sutures were applied, the silk was fragmented and contained lymphocytic foci and young collagenous tissue. Under high power magnification, cross-striation of the graft myofibrils could not be clearly or with surety identified, while those of the ventricle were more easily manifest. In the Group 2 experiments, the histologic picture was similar to that described except for considerable scar tissue in the ventricle corresponding to the area of deficient blood supply. In the Group 3 experiments, firm union was demonstrable be-

tween both walls of the ventricle and the interposed auricular graft. In all three types of experiments, where two auricular grafts were placed in different sites on the ventricle, the histologic picture was similar in both areas.

DISCUSSION

It is conceivable that several uses can be found for an autoplastic graft in cardiovascular surgery. Such a graft may be used for the repair and strengthening of infarcted or ischemic areas of ventricular myocardium which might otherwise rupture or undergo aneurysmal dilatation. It may also be used as a hemostatic agent or reparative graft for application to cardiac wounds of traumatic origin or elective ventricular incisions as those designed for valvulopiasty. Whereas isografts have failed to take because of tissue incompatibility, autogenous grafts have been proved to provide firm and secure union.

SUMMARY

1. In eighteen dogs the auricular appendage has been autogenously grafted onto the ventricular myocardium according to three types of plans: (a) the normal ventricle, (b) the ventricle with an area of deficient blood supply, and (c) the ventricle from which a segment of tissue has been excised.

2. After a ten-week postoperative observation period, the majority of the grafts, although markedly diminished in size, showed firm and continuous union with the subjacent recipient ventricular myocardium.

REFERENCES

1. Burdette, W. J.: Removal of the Auricular Appendage in the Dog, Surg., Gynec. & Obst. **89**: 623, 1949.
2. Beck, Claude S.: Principles Underlying Operative Approach to the Treatment of Myocardial Ischemia, Ann. Surg. **118**: 788, 1943.

TRAUMATIC BRONCHIAL RUPTURE WITH PLASTIC REPAIR

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RUPTURE of a bronchus is an unusual result of nonpenetrating trauma to the chest. The diagnosis has been made only rarely during the period immediately following the trauma. If the patient survives this period, the rupture or fracture of the bronchus usually heals, often resulting in either complete occlusion or a stricture of the bronchus.

The following two patients, both of whom recovered, are reported to emphasize the difference in treatment of a traumatic bronchial occlusion in contrast to a traumatic bronchial stricture, and to call attention to the diagnosis and pathology.

CASE 1.—R. L., a 29-year-old white man, was admitted to Baylor Hospital on Nov. 21, 1947, with a history of a chronic cough for ten years and an episode of severe hemoptysis six weeks previously. The patient had sustained a chest injury with multiple rib fractures in an automobile accident in 1937. He was kept in an oxygen tent for sixteen days, but eventually made his recovery. Since that time, however, the patient had had a chronic productive cough, frequent colds, and respiratory tract infections. He had pneumonia in 1944 which was followed by a persistent wheeze in the right side of the chest. In October, 1947, he had a severe hemoptysis of about one pint of bright red blood. Physical examination was negative except for the chest findings. The right side of the chest was dull to percussion, and breath sounds were markedly diminished. A wheeze, present during both inspiration and expiration, was audible over the right lung field. Roentgenograms of the chest revealed a total atelectasis of the right lung with marked displacement of the heart and mediastinum into the right side of the chest (Fig. 1 *A*). Multiple cavities and areas of pneumonitis were visualized in the right lung. The left lung appeared normal except for overexpansion. Bronchoscopy revealed the trachea and carina to be displaced to the right. Just lateral to the carina, the right bronchus was partially occluded by a stricture, the opening in which was about one millimeter in diameter. Bronchography revealed severe saccular and cystic bronchiectasis of the right lung and a normal left lung (Fig. 1 *B*). A diagnosis of pulmonary suppuration secondary to a traumatic bronchial stricture was made and a pneumonectomy was advised.

On Nov. 25, 1947, a right total pneumonectomy was done. The lung was shrunken and airless. A true right main bronchus could not be found, there being only a bulbous dilatation of the bronchus alongside the trachea from which the secondary bronchi arose directly.

Pathologic examination of the lung revealed a severe saccular bronchiectasis, chronic pneumonitis, and atelectasis.

The patient's course postoperatively was uneventful, and subsequent examinations revealed a good result.

(This case is included through the courtesy of Dr. Robert R. Shaw who cared for the patient.)

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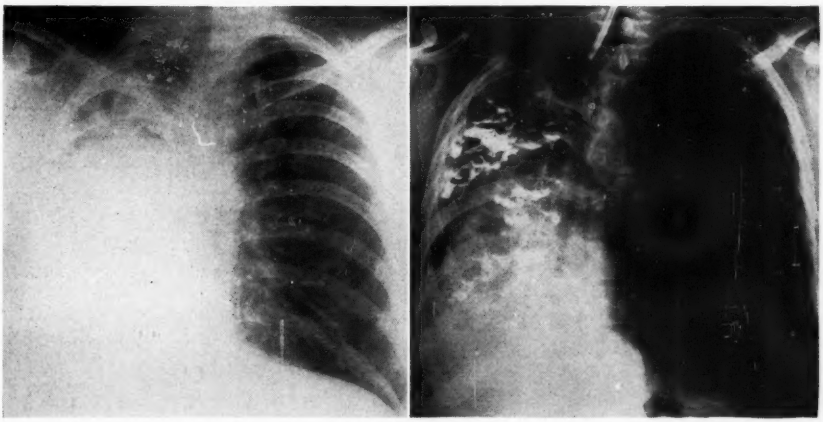
Comment: The patient sustained a bronchial rupture secondary to severe nonpenetrating chest trauma. The fractured bronchus healed but with stricture formation. Pulmonary suppuration and severe bronchiectasis developed distal to the stricture. Pneumonectomy was necessary to relieve the patient of his pulmonary suppuration.

CASE 2.—C. M., a 22-year-old white man, an oil field worker, sustained a severe chest injury on May 24, 1949. While unloading heavy drill pipe from a truck, he slipped and fell, the heavy pipe striking and rolling across his chest and neck. He was taken to the local hospital where an examination revealed a mediastinal emphysema and a right tension pneumothorax. There were no rib fractures. The air extended rapidly into the neck, the face, the arms, and chest wall. The patient's condition became critical rapidly. A mediastinotomy through a suprasternal or cervical approach was done immediately, and a needle was inserted into the chest for the release of the air under tension in both the mediastinum and pleural cavity. The patient's condition improved immediately. The needle in the chest was later replaced by a catheter inserted through the right second interspace anteriorly and attached to a water seal system.



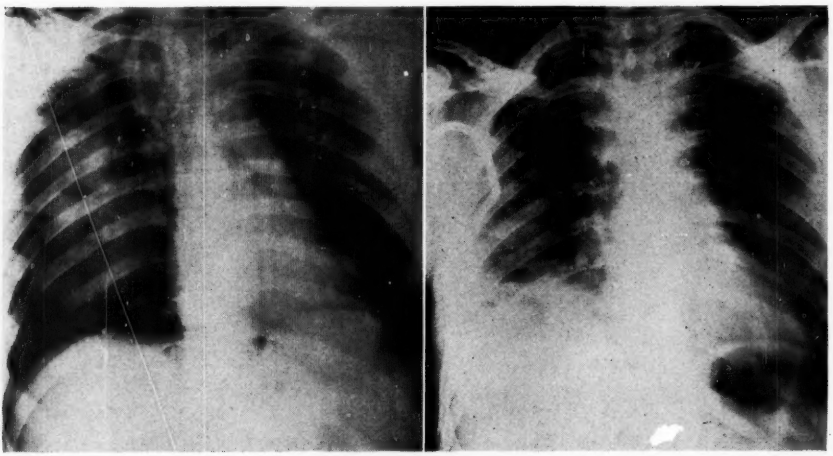
A. *B.*

Fig. 1.—(Case 1) *A*, Roentgenogram of the chest illustrating atelectasis of the right lung with displacement of the heart and mediastinum into the right side of the chest and overexpansion of the left lung. *B*, Bronchogram illustrating severe saccular and cystic bronchiectasis in the atelectatic right lung.

The patient was first seen by the author in consultation on May 25, 1949, the day following the accident. At that time, the diagnosis of a bronchial rupture was entertained because of the sudden, severe mediastinal emphysema and tension pneumothorax which occurred immediately following the accident in the absence of rib fractures. The diagnosis was discounted, however, on the basis of prompt re-expansion of the lung. Further diagnostic procedures, such as bronchoscopy, were not done in view of the patient's satisfactory course. The leak of air stopped and the catheter was removed from the chest a few days after the accident (Fig. 2).

The patient was again referred to the author on July 1, 1949, about seven weeks later, because the attending physician was not satisfied with his progress. The patient stated that he had noted a wheeze in his "throat" up to one week previously when it had suddenly disappeared. He had also noted increasing shortness of breath and chest pain. Serial roentgenograms of his chest revealed good expansion of his right lung until June

29, 1949 (Fig. 3), when the right lung began to shrink in volume. A roentgenogram of the chest made on July 1 (Fig. 4) revealed further shrinkage of the right lung, although it still contained some air. Bronchoscopy and bronchography (Fig. 4) done on that date revealed a complete occlusion of the right main bronchus at the level of the carina. A diagnosis of a bronchial rupture followed by healing with occlusion was made and surgery advised. The patient was admitted to Baylor Hospital on July 2, 1949, where another roentgenogram of the chest revealed complete atelectasis of the right lung and a shift of the mediastinum to the right side (Fig. 4). Dyspnea and chest pain were severe and were due to a high negative intrathoracic pressure. It was decided to save the patient's right lung by a plastic reconstruction of the right main bronchus.



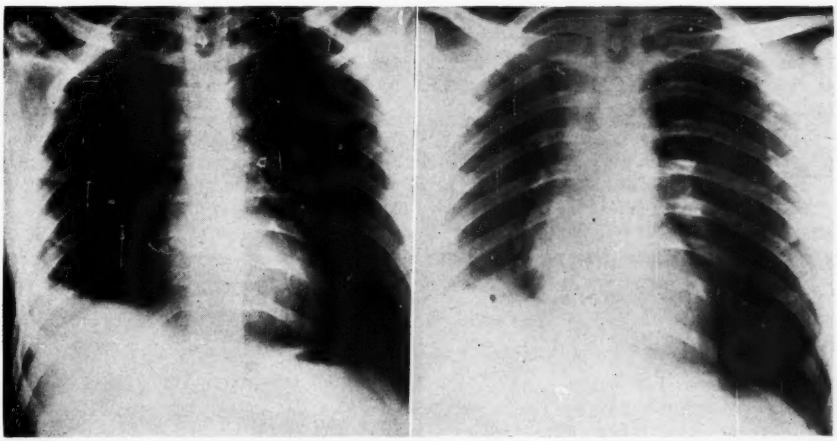
A. *B.*

Fig. 2.—(Case 2) Roentgenograms of the chest: *A*, Made on the day of the accident illustrating the right tension pneumothorax and mediastinal emphysema. *B*, Made two days after the accident illustrating almost complete expansion of the right lung. Intrapleural catheter still in place.

The patient was operated upon on July 2, 1949. The anesthetic was administered through an intratracheal tube inserted into the left main bronchus. A thoracotomy through the right fifth rib bed was done. The right lung was completely atelectatic and there were numerous adhesions between the visceral and parietal pleurae. These adhesions were divided and the lung retracted anteriorly and medially. The mediastinal pleura overlying the trachea and the right main bronchus was incised. The scar in the right main bronchus was readily palpated just below the carina. The membranous portion of the lower trachea and right main bronchus was incised, the incision being carried through the area of bronchial occlusion posteriorly. On incising the bronchus distal to the occlusion, a large amount of thick, gelatinous material exuded from the lumen. Heavy scar was present in the bronchial wall medially and anteriorly. The mucosa was undermined for a short distance and using fine cotton was sutured over the fibrous scar in a transverse direction. An ovoid defect in the membranous portion of the lower trachea and upper bronchus remained. A dermal graft measuring 2 by 4 cm. was then taken from the skin of the back. The epidermis and subcutaneous fat were removed. The dermal surface was placed over the defect in the trachea and bronchus and sutured in place by means of two rows of interrupted fine cotton mattress sutures. The intratracheal tube which had been in the left main bronchus was then withdrawn into the trachea. Under positive pressure the right lung expanded, but air leaked around the dermal graft. After

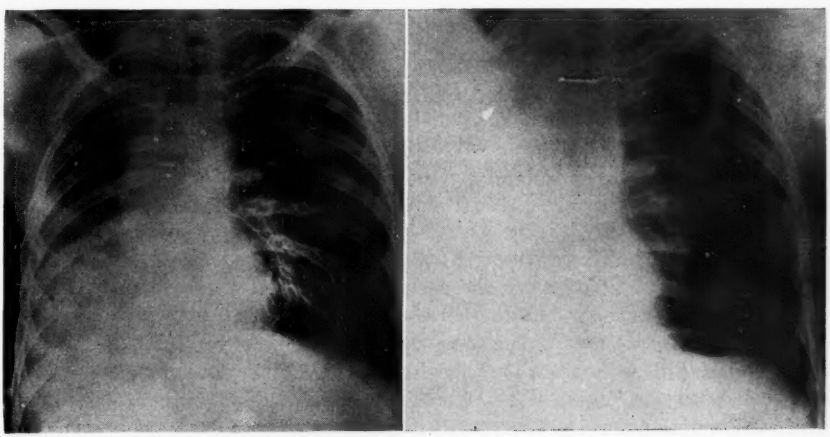
suturing the mediastinal pleura over the graft the leak of air stopped. A large catheter was introduced into the pleural cavity through the eighth interspace in the posterior axillary line, and the chest was closed in the usual manner.

The postoperative course was entirely uneventful. Early complete expansion of the right lung was obtained, and the catheter was removed in twenty-four hours. The patient left the hospital on the seventh postoperative day.



A. *B.*

Fig. 3.—Roentgenogram of the chest (Case 2): *A*, Made on June 20, 1949 (four weeks after the accident), revealing good expansion of the right lung and the mediastinum in normal position. *B*, Made on June 29, 1949 (six weeks after the accident), illustrating beginning shrinkage of the right lung and shift of the mediastinum to the right.

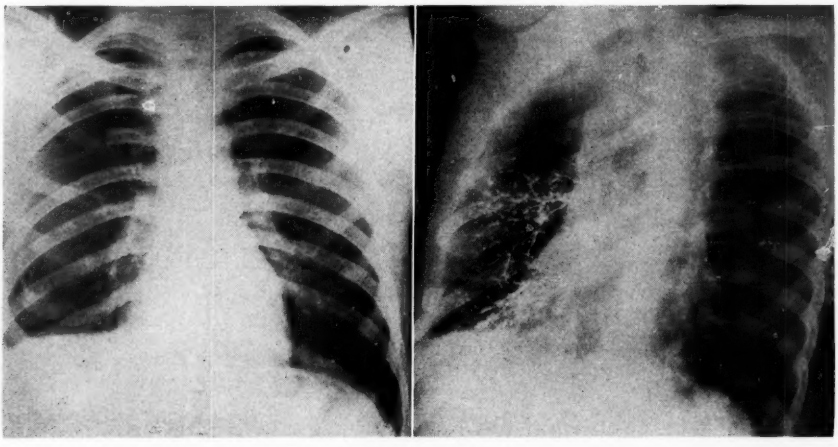


A. *B.*

Fig. 4.—*A*, Bronchogram (Case 2) done July 1, 1949, revealing complete occlusion of the right main bronchus and further shrinkage of the right lung as compared with roentgenogram made on June 29, 1949. *B*, Roentgenogram of the chest made on July 2, 1949, in which the right lung is totally atelectatic and the mediastinum shifted markedly to the right.

The patient returned one month later, on Aug. 1, 1949, at which time a roentgenogram of the chest revealed complete expansion of the right lung (Fig. 5). Bronchoscopic examination revealed a patent right main bronchus with an intact mucosa. The only evidence of the occlusion was a narrowing of the bronchial lumen about one-half centimeter distal to the carina. This narrowing was due to the fibrous scar visualized at the time of the plastic repair of the bronchus. The graft site was smooth and covered by bronchial mucosa. A 7 mm. Broyles bronchoscope could be readily passed through the area of narrowing. The bronchial tree appeared normal. Bronchography done on the same day revealed a normal bronchial tree with no evidence of dilatation (Fig. 5). There was more apparent narrowing at the site of the previous occlusion than was visualized bronchoscopically. This narrowing is due to the scar in the bronchial wall medially and anteriorly which was not disturbed at the time of the plastic repair.

The patient was seen again on Sept. 8, 1949, at which time he stated he had no cough, no wheeze, and no shortness of breath. Physical examination of the chest was negative, and a roentgenogram revealed complete expansion of the right lung. He was still doing well and working nine months after his injury.



A. *B.*

Fig. 5.—*A*, Roentgenogram of the chest made on Aug. 1, 1949 (one month after bronchoplasty), revealing complete expansion of the right lung and the mediastinum in normal position. *B*, Bronchogram one month after bronchoplasty revealing a normal bronchial tree with the exception of some narrowing at the site of the previous occlusion.

Comment: The diagnosis of traumatic bronchial rupture was entertained within twenty-four hours after the accident because of the severe mediastinal emphysema and tension pneumothorax which developed immediately in the absence of rib fractures. The diagnosis was considered unlikely, however, because of rapid re-expansion of the lung. The subsequent shrinkage of the lung which occurred three weeks later culminating in complete atelectasis and associated with a wheeze subjectively again suggested the diagnosis of a rupture of the bronchus. Bronchoscopy and bronchography revealed a complete bronchial occlusion. A bronchoplasty was done by means of a dermal graft with restoration of the bronchial lumen. The lung has completely re-expanded and the patient returned to his occupation as a laborer three months after his injury.

DISCUSSION

Reports in the literature indicate that traumatic rupture of the bronchus occurs rarely and is highly fatal. It is probably true that its infrequent clinical recognition is responsible for these impressions which may be erroneous. Kinsella and Johnsrud[1] in 1947 published a comprehensive review of the literature on traumatic rupture of the bronchus and reported two cases of their own. They found thirty-eight cases. A great many of these ended fatally within a short time following the accident and were usually unrecognized as rupture of the bronchus until autopsy revealed the true condition. A smaller number of patients slowly recovered spontaneously, the exact nature of the condition going unrecognized for months or even years following the injury. The correct diagnosis was made early in a few instances, and all of these patients survived. Including the two cases reported herein, at least eleven more patients have been reported, all of whom recovered.[1, 2, 3, 4, 16, 17] It is anticipated that a larger number of patients will be found with this condition and more will recover completely as a result of earlier diagnosis, modern treatment of the acute traumatic condition involving the chest, and more appropriate late treatment.

The mechanism by which external nonpenetrating trauma produces a rupture of the bronchus is not known, but it is probably similar to that of rupture of the aorta, diaphragm, bowel, or bone due to a nonpenetrating indirect force. It has usually been associated with a crushing type of injury to the chest with or without fractures of the ribs. Schönberg[5] suggested that actual compression of the bronchus occurs between a sudden strong force being applied to a relatively elastic chest wall anteriorly and a rigid spine posteriorly, the lungs being in an expiratory phase with the glottis closed. At any rate a severe shearing force apparently causes the semirigid bronchus to rupture, whereas the softer more elastic vessels and lung may be able to absorb the force and not be damaged.

The point most likely to be torn is the main bronchus either at or within a few centimeters of the carina. Secondary bronchi may be torn. There may be either a partial tear or complete transection of the bronchus. Usually the cartilage is fractured. It is conceivable that complete healing with no residual deformity might occur, but some degree of bronchial stenosis or complete occlusion usually results owing to separation and malalignment of the bronchial ends. Complete occlusion occurred in eight of eleven cases reported since 1947, including the two cases herein reported.[1, 2, 3, 4, 16, 17]

The most common result of rupture of a bronchus appears to be complete occlusion developing in from a few days to a few weeks. Aeration of the lung ceases and the lung remains in a chronic state of true atelectasis without suppuration. Suppuration occurs only if a small opening is present, as in the case of a stricture. This fact has been repeatedly demonstrated clinically and pathologically in the reported cases of bronchial rupture. Apparently complete bronchial occlusion precludes the entrance of bacteria into the atelectatic lung. Krinetski's[6] patient died of pulmonary tuberculosis in the contralateral lung twenty-one years after sustaining a ruptured bronchus with complete bronchial occlusion and atelectasis. The atelectatic lung showed no evidence of either

tuberculosis or suppuration. Hollinger, Zoss, and Johnston report a patient having complete occlusion of the bronchus with atelectasis in whom a pneumonectomy was done three years after the injury. No evidence of infection or pathologic change other than atelectasis of the resected lung was found. The bronchi of the atelectatic lung distal to the occlusion usually contain a homogeneous mucoid material which fills the bronchi but which has not undergone suppuration and can be readily suctioned away.

These clinical and pathologic observations in the case of complete bronchial occlusion are supported by the experimental work of Tannenberg and Pinner.[7] These investigators found that complete obstruction of a main bronchus by ligation in rabbits produced complete atelectasis but no suppuration or bronchial dilatation in those animals observed up to two months. A second factor, infection, was necessary to produce bronchiectasis. It appears from these observations in animals and the human being that a completely obstructed bronchus produces only a true atelectasis of the lung, and that this atelectatic lung can exist for prolonged periods of time without undergoing any important pathologic change. This is significant in the treatment of a patient with complete bronchial occlusion due to trauma. These facts suggest that by means of a bronchoplasty the bronchial lumen can be restored and the collapsed lung will resume function unchanged by the period of collapse.

Associated with the total atelectasis due to bronchial occlusion, there occurs a marked shift of the mediastinal structures, contraction of the hemithorax involved, elevation of the diaphragm, and marked overexpansion of the contralateral lung; the latter leads to emphysema which further diminishes pulmonary function. The extent to which this occurs depends on the age of the patient and the duration of the bronchial occlusion.

DIAGNOSIS

The diagnosis of bronchial rupture may be difficult immediately following the injury, but should be relatively easy later. The possibility of a tear in the bronchus should be kept in mind in a patient who has sustained nonpenetrating trauma to the chest and rapidly develops a tension pneumothorax and/or a mediastinal emphysema. If air continues to escape vigorously through a water seal system over a prolonged period of time, a bronchial tear should be suspected, particularly if the lung fails to expand. Atelectasis persisting or occurring subsequent to early re-expansion of the lung suggests traumatic occlusion of the bronchus. Bronchoscopy done as a therapeutic or diagnostic procedure may establish the diagnosis early. The early diagnosis of a tear in one of the main bronchi depends on keeping the condition in mind and recognizing it by certain findings in a patient with a history of nonpenetrating chest trauma of the crushing type.

After the patient has recovered from the acute phase, the occurrence of persistent total lung collapse suggests occlusion due to a tear in a main bronchus. The patient usually complains of severe pain in the chest and dyspnea due to the attendant high negative intrathoracic pressure. Negative pressures up to 58 cm. of water were observed by Tyson and Lyle.[8] Clerf[9] and Tyson and Lyle[8]

reduced the attendant high negative intrathoracic pressure by inducing pneumothorax, and relieved the patients' pain and dyspnea thereby. During the time that the bronchus is becoming occluded or a stricture is forming the patient will complain or cough and wheeze. Bronchoscopy and bronchography are indicated to establish the diagnosis as well as being important in excluding the nontraumatic types of bronchial obstruction. This is particularly true if the patient is seen for the first time several months or years after a traumatic episode. Bronchoscopy and bronchography will also serve to differentiate the opacity of a nonaerated lung from that due to an organized hemothorax.

TREATMENT

The treatment of bronchial tears has not been standardized. The objectives of treatment of traumatic rupture of the bronchus should be: (1) the institution of appropriate measures to aid the patient in recovery from the immediate effects of the injury, (2) the restoration of normal bronchial patency and the prevention of pulmonary suppuration, and (3) the restoration of normal or close to normal lung function.

Immediate Treatment: Because of a rapid and large leak of air from the bronchial tear, tension pneumothorax and mediastinal emphysema are common within a few minutes or hours of the injury. Decompression of the tension pneumothorax and a suprasternal mediastinotomy may be lifesaving measures in the relief obtained from the intrapleural and intramediastinal tension. Oxygen, blood, and plasma, and the usual procedures in the management of severe thoracic injuries, such as repeated intratracheal suction and paravertebral block of the involved segments, should be utilized. A thoracotomy may be indicated if severe bleeding is evident. In all such cases in which a thoracotomy is done for another indication such as bleeding from a large pulmonary vessel, the bronchi should be explored for a tear. Bronchoscopy may be indicated due to intrabronchial accumulation of blood or if a bronchial tear is suspected. The use of bronchoscopy will depend on the patient's course. If this is favorable, conservative measures may be persisted in. However, if the patient's course is unfavorable (all of the above measures having been applied) and a bronchial tear is suspected, immediate bronchoscopy and thoracotomy may be indicated. During such an emergency procedure the bronchial tear should be sutured or, if this is impossible due to loss of bronchial substance, a bronchoplasty using a dermal graft or fascia may be accomplished. Mackler[10] describes a case in which a bronchial tear accompanying a penetrating chest wound was suspected. Intrapleural decompression by means of a catheter failed to relieve the patient of anoxia and dyspnea. Air continued to bubble vigorously through the water seal system and the lung did not expand. The patient died in forty-eight hours. Mackler admits that thoracotomy and suture of the bronchial tear were indicated. Sanger[11] successfully performed thoracotomy and suture of bronchial tears due to penetrating wounds in two patients. Lester[16] successfully sutured a torn and separated left main bronchus in a patient two weeks after his injury. Scannell[17] recently reported the suture of an elliptical laceration of the right main bronchus three hours after the accident occurred. Shaw[18] sutured a bronchial tear six hours after a nonpenetrating chest injury.

Prevention of Suppuration and Restoration of Lung Function: As soon as the patient has recovered from the acute phase of the injury a suspected diagnosis of bronchial rupture may be proved by means of bronchoscopy. If the diagnosis has already been proved, one may adopt a course of watchful waiting or advise early surgery with bronchoplasty in mind. It is conceivable that simple bronchial rupture with restoration of a good bronchial lumen may still be possible at this stage. It is impossible to state at this time which patients will develop a stricture and which a complete occlusion, but that one or the other will develop is highly probable.

Bronchoscopic dilatation is ineffective in forestalling the development of a traumatic stricture or complete occlusion. Jones and Vinson[12] demonstrated a bronchial rupture about one month after injury by means of bronchoscopy, but considered dilatation of the healing bronchial stricture inadvisable. Hollinger and associates[3] found repeated dilatation of a bronchial stricture five weeks after the initial injury to the ineffectual in preventing a complete occlusion from developing. Loeffler and Nager[13] likewise treated their case of bronchial stricture, the result of an injury sustained six months previously, by repeated bronchoscopic dilatations and electrocoagulation with little benefit. The failure of dilatation is understandable on the basis of the pathologic picture, the bronchial cartilage usually being fractured. Griffith[2] attempted repeated bronchoscopic dilatation of a traumatic stricture of the bronchus three months after the initial injury without benefit. His findings indicated a stricture composed only of mucosa being largely due to loss of cartilaginous support, overriding, and malalignment of the two ends.

Pulmonary suppuration with subsequent loss of the lung (Case 1) is a certainty if a stricture develops. Complete loss of function of the involved lung, overexpansion, and emphysema of the contralateral lung with diminished pulmonary function are the results of complete bronchial occlusion.

Once the diagnosis of bronchial rupture has been proved it would seem prudent to proceed with a bronchoplastic procedure before pulmonary suppuration develops and before irreversible changes in the contralateral lung occurs (Case 2). The only reason for deferring surgery at this time would be the belief that the bronchial tear would heal without stricture or occlusion. This result has not been reported in the literature, although it might conceivably occur.

If the patient is seen late, months or years after the initial injury, bronchoscopy and bronchography will reveal the presence of a stricture or occlusion. If pulmonary suppuration is present, a pulmonary resection is indicated. A bronchoplasty is indicated for a complete occlusion. Pneumonectomy is not indicated unless pulmonary suppuration is present. As indicated by Case 2 and the discussion presented above, the atelectatic lung will re-expand and resume function if the bronchial lumen is restored, even years after the initial injury, because during this time it has not undergone any important pathologic change.

The broad term of bronchoplasty requires definition. In recent years various plastic procedures have been utilized to reconstruct the bronchial and tracheal lumen. Belsey,[14] in 1946, reported the successful reconstruction of

practically the entire intrathoracic portion of trachea (excised for tumor) by means of a free graft of fascia sutured over a spiral of stainless steel wire. In 1949, Gebauer[15] published a report of eight successful dermal grafts threaded with stainless steel wire applied to the trachea and bronchi for tuberculous stenosis. Lester[16] recently successfully sutured a torn and separated left main bronchus in a patient two weeks after his injury. Griffith[2] successfully treated a patient with a traumatic stricture of the bronchus by excision and end-to-end anastomosis. These reports and Case 2 herein reported illustrate the feasibility of plastic procedures to reconstruct the tracheal or bronchial lumen in man.

SUMMARY

Rupture of a bronchus may result from nonpenetrating trauma to the chest. The diagnosis has been made only rarely during the period immediately following the trauma. If the patient survives this period, the rupture usually heals with either complete occlusion or a stricture of the bronchus. Early diagnosis, the modern treatment of chest injuries, and bronchial reconstruction by means of suture or bronchoplastic procedures will reduce the mortality, restore lung function, and prevent pulmonary suppuration. In the case of a complete bronchial occlusion the collapsed lung can be saved by a bronchoplasty, regardless of the duration. Pneumonectomy is indicated only for proved pulmonary suppuration distal to a traumatic bronchial stricture.

REFERENCES

1. Kinsella, T. J., and Johnsrud, L. W.: Traumatic Rupture of the Bronchus, J. THORACIC SURG. **16**: 571, 1947.
2. Griffith, J. L.: Fracture of the Bronchus, Thorax **4**: 105, 1949.
3. Hollinger, P. H., Zoss, A. R., and Johnston, K. C.: Rupture of Bronchus Due to External Chest Trauma: Report of Three Cases With Recovery, Laryngoscope **53**: 817, 1948.
4. Hodes, P. J., Johnson, J., and Atkins, J. P.: Traumatic Bronchial Rupture With Occlusion, Am. J. Roentgenology **60**: 448, 1948.
5. Schönberg, S.: Bronchialrupturen bei Thoraxkompression, Berl. klin. Wchnschr. **49**: 2218, 1912.
6. Krinetski, S. L.: Zur Kasuistik siner vollstadigen Zarreissung des Rachten Luftrchrenastes Virchows Arch. f. path. Anat. **266**: 815, 1928.
7. Tannenberg, J., and Pinner, H.: Atelectasis and Bronchiectasis, J. THORACIC SURG. **11**: 571, 1942.
8. Tyson, M. D., and Lyle, J. S.: Traumatic Obstruction of Main Bronchus, New England J. Med. **228**: 192, 1942.
9. Clerf, L. R.: Rupture of Main Bronchus From External Injury, Surgery **7**: 276, 1940.
10. Mackler, S. A.: Experience With Thoracic and Thoraco-abdominal Wounds in an Evacuation Hospital, J. THORACIC SURG. **16**: 538, 1947.
11. Sanger, P. W.: Evacuation Hospital Experiences With War Wounds and Injuries of the Chest, Am. Surg. **122**: 147, 1945.
12. Jones, F. W., and Vinson, P. P.: Nonfatal Rupture of the Left Main Bronchus From External Trauma, Surgery **5**: 229, 1939.
13. Loeffler, L., and Nager, F.: Ueber Traumatische Bronchostenose und ihrs Behandlung, Schweiz. Med. Wchnschr. **71**: 293-97, 1941.
14. Belsey, R.: Stainless Steel Wire Suture Technique in Thoracic Surgery, Thorax **1**: 29, 1946.
15. Gebauer, P. W.: The Use of Dermal Grafts for Tuberculous Stenosis of the Trachea and Bronchi, Hawaii M. J. **413**: 1949; Plastic Reconstruction of Tuberculous Bronchostenosis With Dermal Grafts, J. Thoracic Surg. **19**: 604, 1950.
16. Lester, R. T.: Personal communication.
17. Scannell, J. G.: Rupture of the Bronchus Following Closed Injury to the Chest, Ann. Surg. **133**: 127, 1951.
18. Shaw, R. R.: Personal communication.

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The Thirty-second Annual Meeting of the American Association for Thoracic Surgery will be held May 8, 9, and 10, 1952, in Dallas, Texas. Headquarters will be at the Hotel Baker. The Adolphus Hotel and the Southland Hotel are near by and will also have suitable accommodations.

Request for Hotel Reservations: These should be mailed directly to S. Hugh White, Room Reservation Manager, Hotel Baker, Dallas 1, Texas.

Abstracts for Papers: Abstracts for the presentation of papers at the 1952 Meeting must be received for consideration by the Program Committee on or before Jan. 1, 1952. These abstracts should be submitted in *quadruplicate* and addressed to the Secretary of the Association, Dr. Paul C. Samson, 2938 McClure St. Oakland 9, Calif. They should not be sent to the Editor. Abstracts received after Jan. 1, 1952, will not be considerd.

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